OBSERVATIONAL STUDY OF FOURNIER'S GANGRENE AND USEFULNESS OF FOURNIER'S GANGRENE SEVERITY INDEX IN PREDICTING THE OUTCOME

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CHENNAI

CERTIFICATE

This is to certify that the dissertation entitled **"Observational study of Fournier's Gangrene and Usefulness of Fournier's Gangrene severity index in predicting the outcome "** is the bonafide work done by **Dr. P.NAVEEN.,** Post Graduate student (2010 – 2013) in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under my direct guidance and supervision, in partial fulfilment of the regulations of The Tamil Nadu Dr. M.G.R Medical University, Chennai for the award of M.S., Degree (General Surgery) Branch - I, Examination to be held in April 2013.

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DECLARATION

I, solemnly declare that this dissertation titled "**Observational study of Fournier's Gangrene and Usefulness of Fournier's Gangrene severity index in predicting the outcome**" is a bonafide work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the guidance and supervision of my unit chief **Prof. A. RAJENDRAN, M.S.,** and my Head of the Department **Prof. P. DARWIN, M.S.,**

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Place : Chennai

Date : December 2013 **DR. P.NAVEEN**

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It is my earnest duty to thank my parents without whom accomplishing this task would have been impossible.

I am extremely thankful to my patients who consented and participated to make this study possible.

CONTENTS

INTRODUCTION

Fournier's gangrene (FG) is an acute, rapidly progressive and potentially fatal, necrotizing fasciitis of infective etiology affecting the scrotum and penis, perineal and perianal regions. It leads to the thrombotic occlusion of small subcutaneous vessels, resulting in the development of gangrene of the overlying skin. It may extend to the medial aspects of thigh and Anterior abdominal wall and can go onto chest wall.

Bauriene [(1)] first reported this form of disease, King Herod the Great of Judaea was found to be affected by the disease and he was probably a diabetic. . He described it as a rapidly progressive necrosis of male external genitalia of idiopathic nature. The genuine and more detailed account of this condition came from a dermatologist in France **Dr. Jean A. Fournier in 1883.**

The disease affects all groups of age and there are many etiological and predisposing factors described, this is more common in patients with immunosuppressive disorder like diabetes mellitus, malignancy and chronic alcoholism.

The basic treatment involves active resuscitation, immediate excision of all gangrenous and necrotic tissues, to limit the spread of infection, antibiotics administration and reconstruction at a later date to achieve skin cover.

Underlying co-morbidities and the rapid spread of infection contribute to the mortality in this condition which ranges from 16-50% in many series in spite of the availability of modern critical care units and advanced ICUs.

Laor et al. developed the **Fourniere's Gangrene Severity Index (FGSI)** to stratify risk in these patients. FGSI is a numerical score obtained from a combination of physiological hospital admission parameters that include **heart rate, respiratory rate, temperature, serum sodium in meq/l, serum potassium in meq/l, creatinine, white cell count, hematocrit and serum bicarbonate**. Laor proposed that an FGSI above nine is both sensitive and specific as a mortality predictor in FG patients.

AIM & OBJECTIVES

1. To study the incidence and prevalence of Fournier's gangrene.

2. To assess predisposing factors, etiological factors and duration of symptoms of Fournier's gangrene.

3. To analyze the need for repeated surgical debridement and the interval between them will be recorded and assessed.

4. To assess the various factors influencing the final outcome.

4. To ascertain the usefulness of Fournier's gangrene severity index in predicting the mortality and morbidity.

MATERIALS AND METHODS

STUDY DESIGN

Observational study

SAMPLE SIZE

35 cases of Fournier's gangrene

POPULATION

Surgical wards at Govt. Stanley Hospital

PERIOD OF STUDY

01.01.2012 TO 31.12.2012

INCLUSION CRITERIA:

All patients diagnosed to have Fournier's gangrene between the ages 20 to 80.

Both sexes are included.

EXCLUSION CRITERIA

Age < 20 years

Patients in whom, the initial wound debridement is done in other institutions and referred here later.

METHODS

1. Patients of Fournier's gangrene admitted to Emergency surgical ward were included in the study after obtaining informed consent. Data's of those patients were drawn and analyzed statistically.

The following data were collected using proforma

Detailed history regarding

- onset
- Duration of symptoms
- Progress of disease
- Predisposing factors
- Etiological factors

General condition of the patient

Vital parameters

- Heart rate
- Respiratory rate
- Temperature

Basic Investigations

- Complete blood count
- Sugar, urea, creatinine
- Serum sodium, potassium and bicarbonate.

Wound culture & sensitivity

Based on hospital admission parameters Fournier's gangrene severity index score is calculated and score was obtained to each patient.

3. All patients were treated routinely by immediate surgical debridement and administration of broad spectrum antibiotics. This was changed once the antibiogram report is obtained from sample sent for wound culture and sensitivity.

4. All patients were followed up during the hospital stay and details regarding no. of debridements were noted.

5. The need for diversion procedure to eliminate fecal and urinary contamination in extensive wounds is observed and analyzed.

6. Once the infection is controlled the method adopted in reconstruction of the wound is noted.

7. The final outcome of the patient is compared with the clinical score and its usefulness in detecting the morbidity and mortality will be analyzed.

REVIEW OF LITERATURE

Fournier's gangrene is a rather uncommon, aggressively spreading infection that initially affects the genitalia and perianal regions with progression to the abdomen and thoracic wall. The hall mark of the disease is gangrene of skin and subcutaneous tissues due to thrombotic occlusion of subcutaneous vessels caused by necrotizing fasciitis.

HISTORICAL BACKGROUND

Hippocrates and other medical forefathers had described this scrotal condition in their writings, surprisingly; this disease has not been recorded until the later part of 1700 s.

The first report of gangrene of the penis was made in 1764 by **Baurienne**. Baurienne originally described an idiopathic, rapidly progressive soft-tissue necrotizing process that led to gangrene of the male genitalia.

The gangrene process in genitalia was again described in 1873 by **Avicennaa Ibn sinha** in his book "The Canon Of Medicine"

The authentic description of the disease was done by J.A.Fournier in 1883, specifically in the scrotum. He described it as **"fulminant gangrene of the penis and scrotum"** based on the findings in five young men with gangrene of the scrotum [2].

The disease was defined with following features:

(1) Sudden onset in a hitherto healthy young man,

(2) Rapid progression to gangrene and

(3) Absence of a definite cause.

Jean A. Fournier

Frank Lamot Meleney (from New York), while in Beijing, China during World War I (1920s) observed a series of cases with extensive skin and subcutaneous tissue necrosis involving extensive areas in the body and found it to be causally associated with streptococcus and termed it "streptococcal gangrene"[3] and the term "necrotizing fasciitis" was coined by Wilson in the year 1952[4]. Fournier's gangrene is otherwise also known as phagedena, periurethral phlegmon and synergistic necrotizing cellulitis[5,6,7].

In the early nineties Sensational medical journalists dramatized the whole process of necrotizing fascitis by associating it with flesh-eating bacteria

and authors suggested that the whole process of necrotizing fasciitis to be due to bacteria s eating away the tissues and termed it as **flesh eating bacteria**. However it was found to be a misnomer because bacteria only causes occlusion of vessels and this in turn results in sloughing of tissues.

A comprehensive and widely accepted definition of Fournier's gangrene was proposed by Smith et al[8] as **'an infective necrotizing fasciitis of the perineal, genital or perianal regions'**.This definition was not restricted to men of all ages like in Fournier's original report, but also included women and children.

RELEVANT ANATOMY

A thorough knowledge of anatomy of perineum , layers of scrotum and penis ,and layers of anterior abdominal wall is required to understand the spread of disease. The spread of Fournier's gangrene is due to the multiple intercommunicating fasciae from the region of perineum to anterior abdomen wall. Infective and necrotic process involves the superficial and deep fascia and the muscles underneath are usually spared. Both debridement initially as well as reconstruction later on are affected by this pattern of spread.

ABDOMEN- SUPERFICIAL LAYERS (SKIN, SUBCUTANEOUS FAT AND FASCIA)

Since this condition is basically the result of infection of the superficial and deep fascia, it is absolutely necessary to understand the anatomy and relationship of the skin and subcutaneous tissue of the abdominal wall and perineum.

The skin above the inguinal ligament is supported by superficial Camper fascia. This is a layer containing fat of variable thickness and superficial vessels that supply the skin .Scarpas fascia is deep to camper fascia and this continues below as colles fascia in perineum and dartos fascia in scrotum (Figure 1).

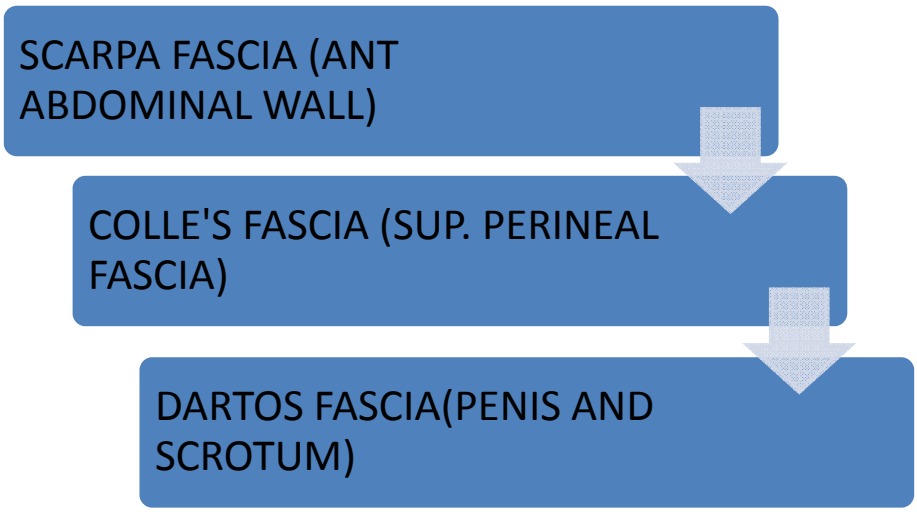
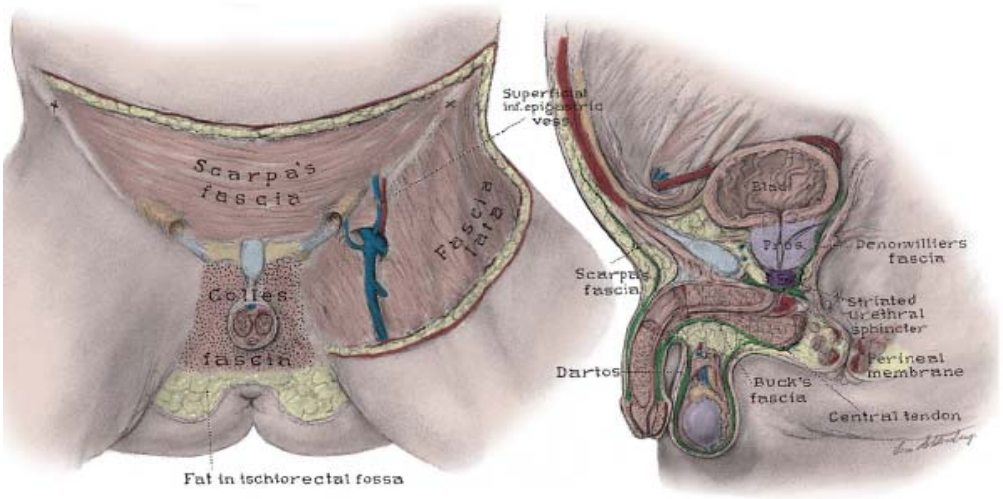


Figure 1



PERINEUM

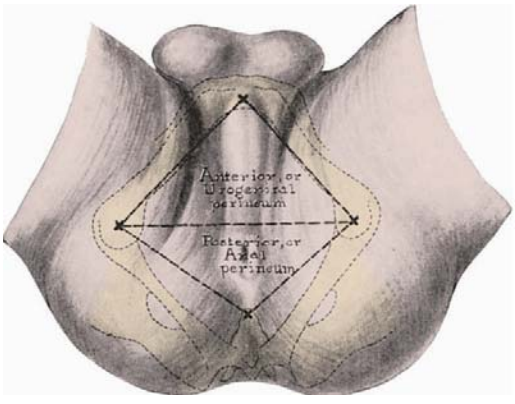
In the anatomic (upright or erect) position, the perineum is a narrow area of soft parts located between the musculature of the gluteal and thigh areas. With abduction of the thighs, the perineum has a diamond-shaped configuration. The diamond is bordered by the ischiopubic rami and pubic symphysis in its anterior half, and in its posterior half, or anal triangle, boundaries are provided by the inferior border of the gluteus maximus muscle, the ischial tuberosities, the sacrotuberous ligaments, and the coccyx.

An imaginary line connecting the ischial tuberosities subdivides the perineal diamond into two triangles (Figure2)

ANTERIOR (URO GENITAL TRIANGLE)

POSTERIOR (ANAL TRIANGLE)

Figure 2



Layers of the Urogenital Triangle

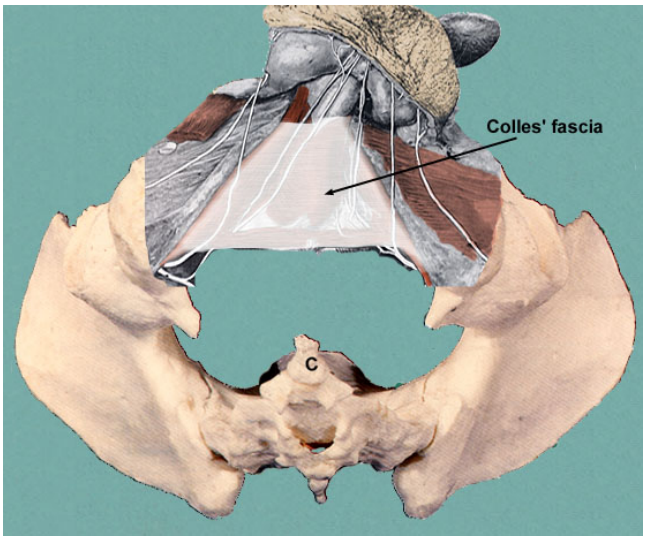
Progressing from superficial to deep (or inferior to superior), the urogenital triangle contains the following layers

Skin and adipose layer: continuous with Camper's fascia of abdomen

Superficial fascia (Colles' fascia) (figure 3**)**

The irregularly membranous, often laminated layer of tissue deep to Camper's fascia is called Colles' fascia. It is the continuum of Scarpa's fascia in abdomen onto the perineum. It is laterally attached to pubic ramus and to the posterior edge of the urogenital musculature posteriorly. This provides an anatomic barrier between the urogenital spaces anteriorly and the ischioanal fossae posteriorly.

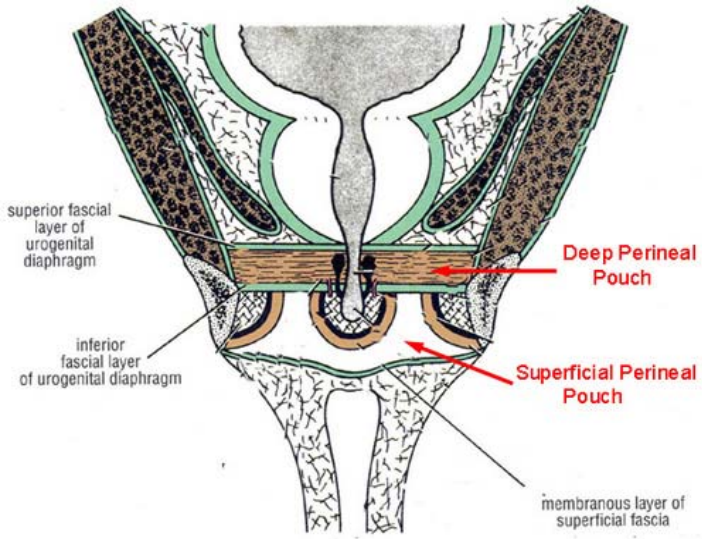
Figure 3



Superficial Perineal Pouch

The superficial perineal pouch includes the space between the fascia of Gallaudet and the perineal membrane. The perineal membrane provides a "roof" for the superficial pouch and a "floor" for the deep pouch (urogenital diaphragm). The perineal membrane is the inferior fascia of the urogenital diaphragm (figure 4).

Figure 4



It is composed of the following structures (from external to internal).

External perineal fascia or muscle fascia of Gallaudet

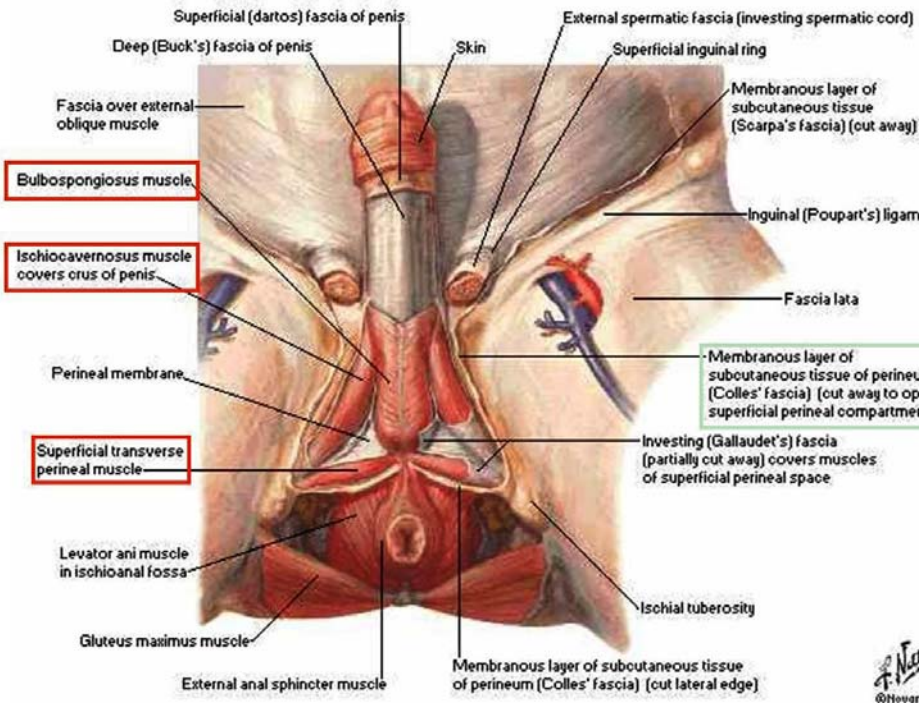
Paired muscles

- Ischiocavernosus
- Bulbospongiosus
- Superficial transverse perineus muscle

OTHERS

- Crura of the Corpora cavernosa of penis.
- Bulb of the penis or Corpora spongiosa.
- In female Right and left vestibular (Bartholin) glands (Figure 5)

Figure 5



The blood supply to the pouch is through Posterior scrotal/labial branches of the perineal branch of the internal pudendal vessels and Transverse perineal branch of the perineal branch of the internal pudendal artery (supplying the superficial transverse perineus muscle and tissue between the bulb and the anus).

The infective process from urogenital tract may track down here and spread to rectum and ischiorectal fossa which are posteriorly and laterally related to the pouch. So the infection from urogenital tract may spread via the superficial perineal pouch to ischiorectal fossa and vice versa.

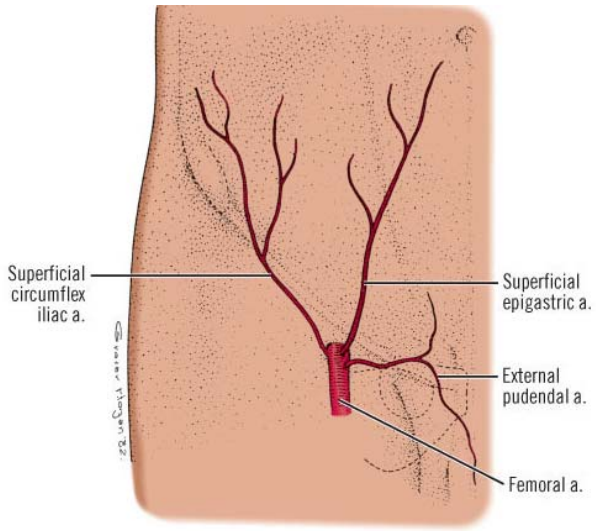
But in majority of cases the infection originating from urogenital tract involves the penis and superficial perineal pouch not extending to the posterior half of perineum. The colles fascia acts as a barrier to limit the spread. The infections from colorectal sources extend from the perianal area to perineum then goes on to involve the anterior abdominal wall without much extension to penis.

There is free space that exists between the external oblique aponuerosis and the Scarpa's fascia. This permits the spread of infection from the perineum onto the anterior abdominal wall..The fascia of abdomen continues in the chest

wall till the clavicle and so the disease process can spread till the chest wall which had initially started in the perineum and scrotum.

Cutaneous blood supply of skin of the anterior abdomen wall and genitalia

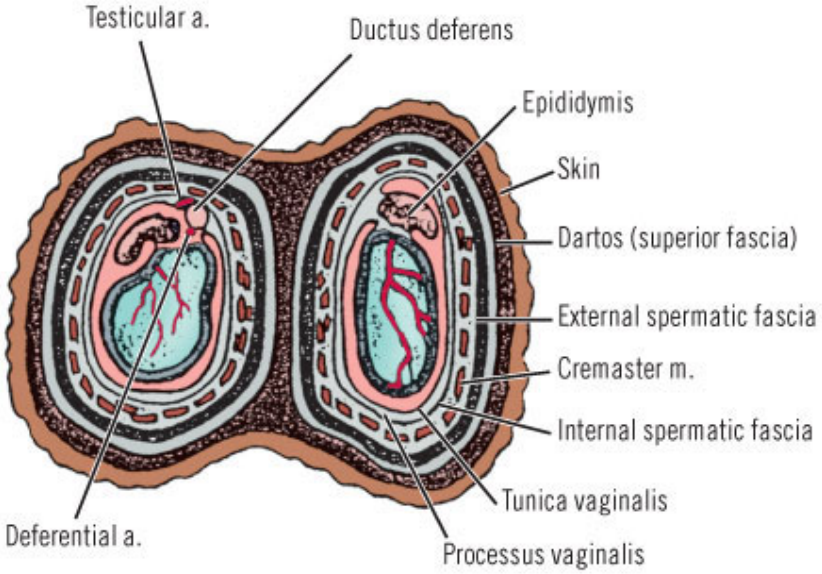
The superficial tissues of the lower anterior abdominal wall are supplied by 3 branches from main femoral artery. These branches, from lateral to medial, are the superficial circumflex iliac artery, the superficial epigastric artery, and the superficial external pudendal artery. The main trunk of these vessels and also the branches of these vessels traverses through the camper fascia and subcutaneous layer of abdominal wall. So it gets obliterated in the process of disease progression. This causes necrosis and sloughing out of tissues anterior abdominal wall (Figure 6).



LAYERS OF SCROTUM

The scrotum houses the testes and the epididymis. It is composed of eight layers that are derived and modified from the six layers of the abdominal wall. Although the layers are continuous, their terminology changes as they pass from abdomen to scrotum(Figure 7) .

Abdominal Wall	Scrotum
Superficial fascia (Camper's and Scarpa's)	Dartos and smooth muscle
External oblique (innominate) fascia	External spermatic fascia
Internal oblique muscle and aponeurosis	Cremasteric fascia and muscle
Transversalis fascia	Internal spermatic fascia
Preperitoneal fat	Preperitoneal fat
Peritoneum	Tunica vaginalis

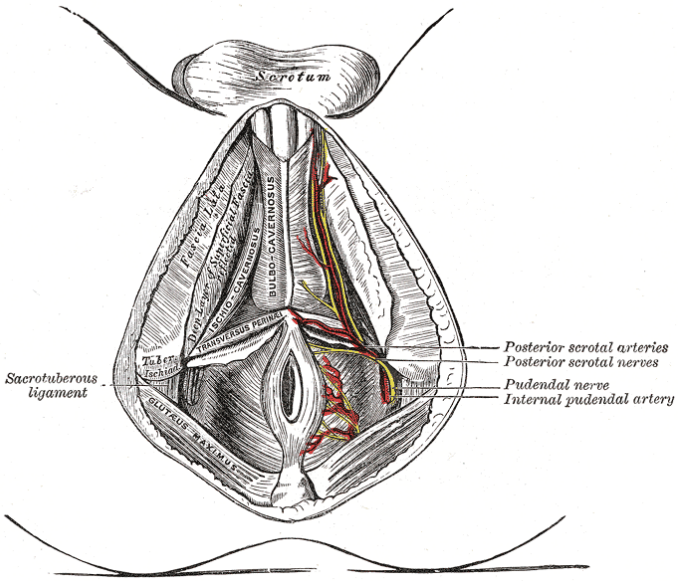


The subcutaneous superficial fascia in the scrotum contains little adipose tissue, this being replaced by smooth muscle that forms the tunica dartos scroti. The attachment of these muscle fibers to the skin forms the rugal folds of the scrotal skin.

The scrotum is well supplied with blood. Branches of the superficial and deep external pudendal arteries supply the anterior part of the scrotum (Figure 8).

Posterior part of scrotum is supplied by internal pudendal artery branch namely posterior scrotal artery. Since the internal pudendal does not travel through the camper fascia the area supplied by it namely the posterior scrotal wall remains intact.

Figure 8



THE PENIS can be divided into three parts: the root, the body, and the glans. The root, or penile bulb, is located within the superficial perineal pouch, which provides fixation and stability. The body is formed by the three spongy erectile anatomic entities: two corpora cavernosa and one corpus spongiosum.

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COVERINGS

(Figure 9)

The **Skin** that covers the penis is thin, with a very thin areolar layer which covers, or is mixed with, the superficial penile fascia. The distal part of the skin forms two anatomic entities, the foreskin (prepuce) and the frenulum.

The ***superficial penile fascia*** is the downward continuation of the fasciae of Camper and Scarpa. It is without an adipose content, but with some smooth muscle fibers, like the dartos tunic of the scrotum.

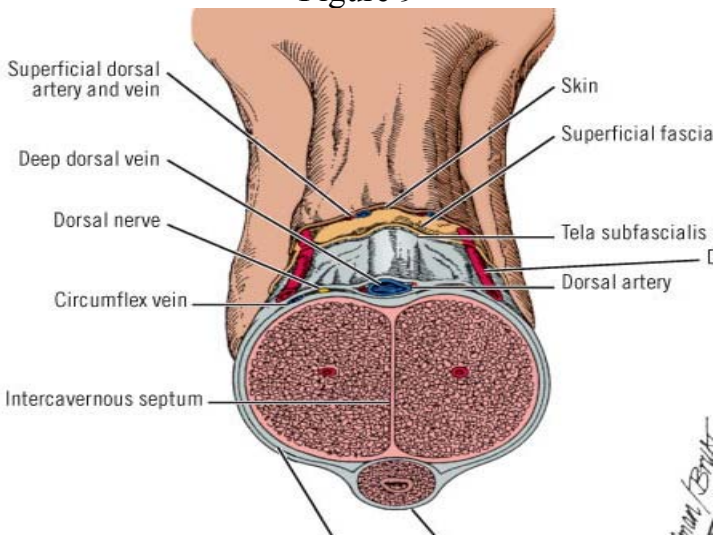
The ***tela subfascialis*** a very thin areolar tissue layer. It occupies the interval between the superficial dartos tunic and Buck's deep fascia over the

extracorporal segments of the cavernous arteries, veins, and nerves. Also in this interval are the bilateral dorsal arteries, dorsal veins, and dorsal nerves.

The ***deep penile fascia*** **(fascia of Buck)** covers the corpora cavernosa and spongiosum. The infection is limited by the buck's fascia.

The ***tunica albuginea*** is a thick white connective tissue matrix formed by two fibrous layers, the outer longitudinal and the inner circular, with little in the way of elastic tissue. It is strongly attached to the overlying fascia of Buck; or perhaps it is better to say that the fascia of Buck is firmly fixed to the tunica albuginea.

Figure 9



The arterial blood supply of the penis is formed by a superficial and a deep system. The external pudendal artery is responsible for the formation of the superficial system; the internal pudendal artery provides the deep system.

Superficial system (Figure 10) originates from the external pudendal artery (from the common femoral artery), which gives origin to a dorsolateral and a ventrolateral branch. These branches supply the skin of the penis.

In deep system (Figure 11) the dorsal artery of penis can be regarded as the terminal continuation of the internal pudendal artery The dorsal artery leaves the urogenital diaphragm by piercing the transverse perineal ligament (the fusion of the superior and inferior fasciae of the diaphragm) and by passing onto the dorsum of the shaft beneath the superficial fascia.

The beneficiaries of the dorsal artery are the corpora cavernosa, the corpus spongiosum, the tunica albuginea, and the urethra which are pierced by branches of the dorsal artery. The dorsal artery also gives off laterally directed circumflex branches which pass to the corpus spongiosum.

FIGURE 10

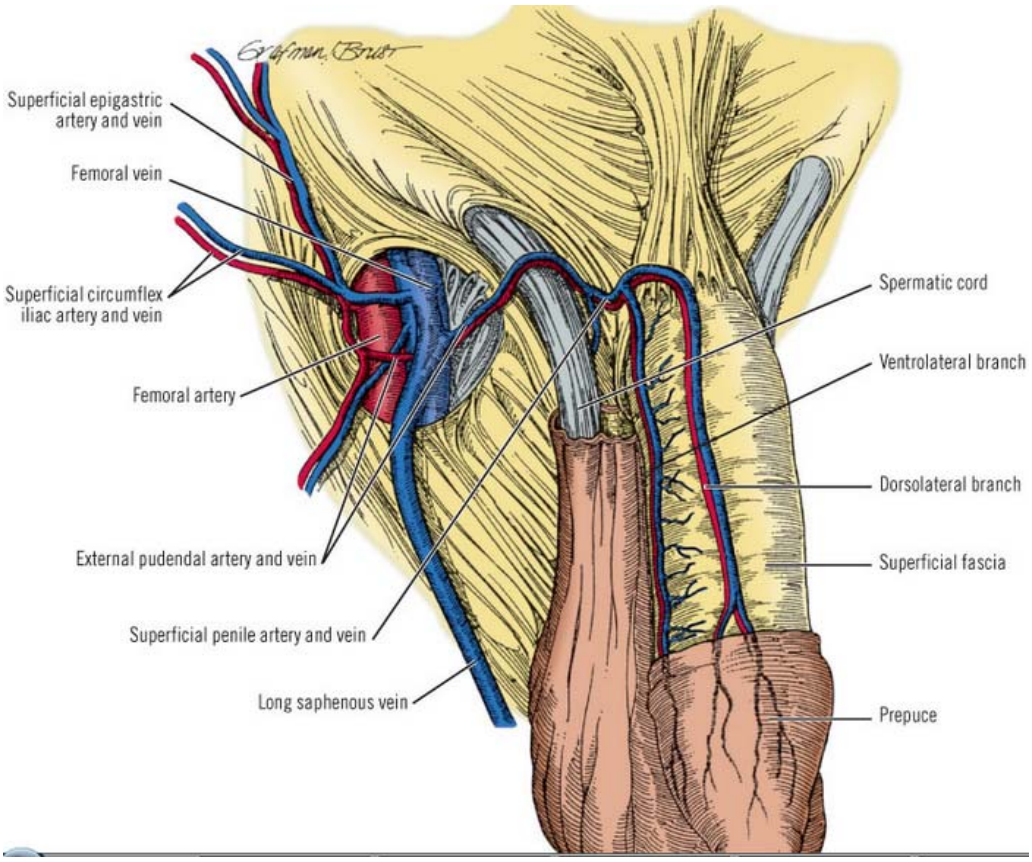
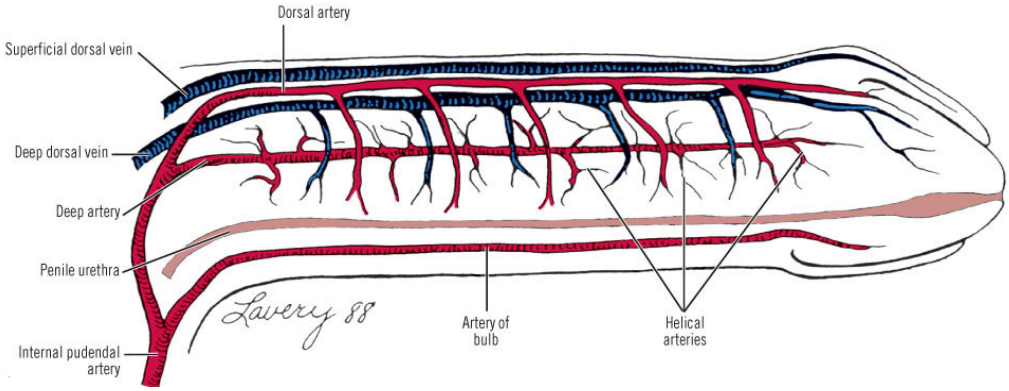
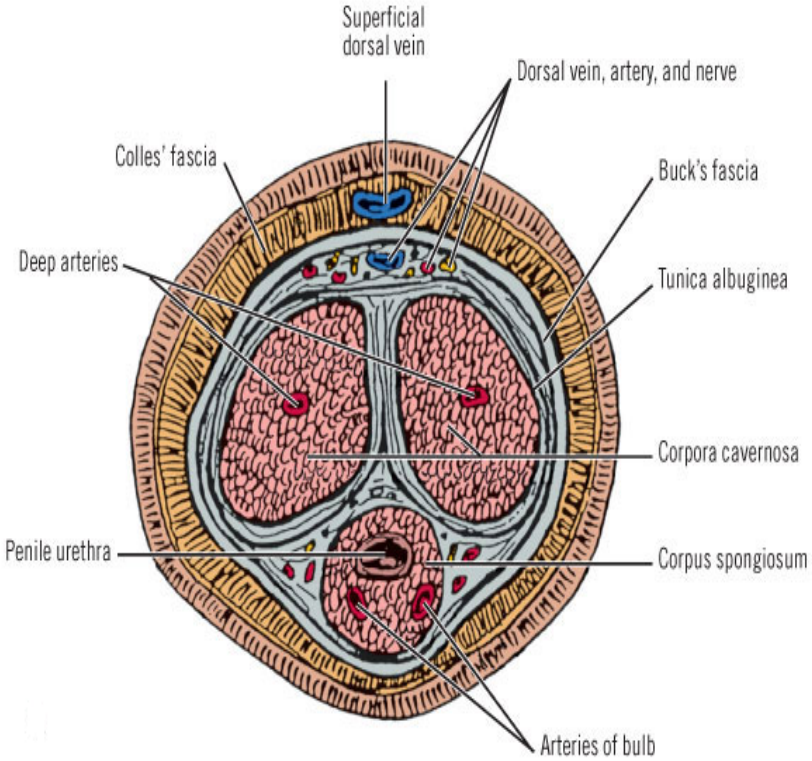

Superficial epigastric artery and vein
Femoral vein
Superficial circumflex iliac artery and vein
Femoral artery
External pudendal artery and vein
Superficial penile artery and vein
Long saphenous vein
Spermatic cord
Ventrolateral branch
Dorsolateral branch
Superficial fascia
Prepuce

FIGURE 11


Dorsal artery
Superficial dorsal vein
Deep dorsal vein
Deep artery
Penile urethra
Internal pudendal artery
Lavery 88
Artery of bulb
Helical arteries



Since the internal pudendal does not travel through the camper fascia the area supplied by it namely the posterior scrotal wall and erectile bodies' remains unaffected so it can be used as a bed to place graft and flaps after the total remission of infective process. When the penis is involved, infective process involves skin and superficial penile fascia gets necrosed and sloughs off while the corpora and urethra are usually intact.

Thrombosis and destruction of the corpara spongiosa and cavernosa has been reported [(22)].Intra abdominal and retroperitoneal infection spread can lead to testicular gangrene [(34)].

EPIDEMIOLOGY

During the period from 1950-1999, 1726 cases was reported in all indexed medical publications. While between 2000 and 2007, 1571 cases have been documented.

This apparent increase in the incidence of this disease may be accounted for by improved identification of the condition when encountered in clinical practice and also because of increased reporting of the disease in medical literature.

Other probable reasons could be an increase in the number of people with predisposing factors such as diabetes, renal failure and immunosuppression (HIV). The internet has an important role to play in disseminating information and creating awareness.

AGE AND SEX

Young men no longer seem to be the only section of the population affected by the disease. The epidemiology of the disease has changed so as to encompass a much larger spectrum of the population ranging from the neonatal age group going upto and including the very elderly.

A review of pediatric literature reveals that of the 55 cases reported, two-thirds have been lesser than 3 months of age [9, 10].

While during the period before 1945, the average age of the patients affected by the disease was about 40 years, 50-60 years seems to be the more commonly affected age group in recent times.

Old age is an indirect predisposing factor to Fournier's gangrene in that poor nutritional status and poor self-care, renders the elderly more susceptible to the disease. The prognosis is also poorer in them.

Genital gangrene has also been reported in women. The disease involves the vulva, perineum, thigh and abdomen as well [11, 12, and 13].

Eke in his large scale meta-analysis, arrived at a male to female ratio of 10:1.

ETHINICITY AND SOCIAL CLASS

While poor socio economic conditions often been quoted as important contributing factors to this disease, supported by a report from Europe stating that the civilized population don't seem to be as commonly affected by Fourneir's gangrene , Some case reports from affluent sections of USA and

Europe beg to differ. They report that the disease affects both the poor and rich sections of the society.

Fournier's gangrene has been reported to be highly prevalent in African countries and South Asian countries.

There are two important etiological sources of sepsis for this condition. One is skin, while the other is colorectal/perineal infections. In the developed world, cutaneous infections are more common source of sepsis and it carries a better survival rate. The morbidity and mortality rates associated with the colorectal source are higher than that encountered with skin. The colorectal route is more frequent portal of entry in the developing countries like ours.

PRE DISPOSING FACTORS

Patients affected by Fournier's gangrene have been found to commonly have an underlying systemic disorder, of which, **DIABETES AND CHRONIC ALCOHOLISM** seem to be the most frequently reported. Chemotherapy, leukemia, liver cell failure , colorectal cancer, carcinoma in other sites with immunosuppression also poses increased risks.

HIV represents a new risk factor in recent times among patients affected by this condition in the developed as well as the developing sections of the

world. In fact, among patients with undiagnosed HIV infection, it has been reported that Fournier's gangrene could be the earliest presentation.

In paraplegic patients bedsore in neighborhood of the scrotum, perineum and perianal regions is also a risk factor.

In all the above mentioned conditions, immnosupression and failure of T cell immunity leading to poor host resistance is the common factor. This followed by decreased oxygen tension and invasion of organisms which under ordinary conditions are commensal to the area, ultimately leads to gangrene [15].

DIABETES AND FOURNIER'S GANGRENE

Among the common systemic diseases encountered among the people in our country, diabetes mellitus has been found to rank high up in the list. True to this fact, 84% of the total number of patients who participated in this study was found to be diabetic.

Diabetes mellitus causes tissue ischaemia by affecting the small blood vessels. It also causes urinary outlet obstruction, thus increasing the incidence of infection of the urinary tract. Other mechanism by which diabetes operates is by causing microvascular disease, affecting phagocytosis and reduction in cellular immunity. All this ultimately results in ischaemia.

ETIOLOGY

Even though it has been emphasized in the original description of this disease that there seems to be no known cause accountable for this condition, further studies have lead to an understanding of the pathological processes that could possibly underlie the occurrence of gangrene of the genital and perineal regions.

The anorectal region, urogenital tract and the skin of the genitalia have been proposed as common sites of the origin of infection which seem to set in motion the necrotizing process.

- **Anorectal causes** - Perianal, gluteal, ischiorectal abscesses;anal fissures; inflammation of the appendix; [17] colonic perforations due to carcinoma, blunt injury and tuberculosis [18, 19], colon and sigmoid diverticulitis and abscess [20].

Urinary tract and External genitalia

- Bulbourethral gland infection,
- Injury to urethra following catheterization or trauma.
- Stricture urethra and infection after manipulation with dialators.
- Renal abscess[21], urethral calculi ,lower urinary tract infection

- Prosthetic penile implants [22], genital jewel piercing [23].

Dermatologic causes –

- Hidradenitis, chronic bedsore, tinea cruris and erypsilea.

- In children - following surgeries like inguinal hernia with strangulation and obstruction, circumcision, urethral manipulation and catheterization for diagnostic purposes , peri-rectal abscesses, rash due to diapers , and burns[24].

- Increased risk is associated with Poor perineal hygiene and the presence of chronically indwelling catheters, for example in paraplegic patients.

- Other surgical causes responsible for Fournier's gangrene – surgery for strangulated femoral and inguinal hernias , Thiersch wiring , banding for pile mass [25], prostatic biopsy [26], femoral puncture for splenic angiography and also rectal foreign bodies[27].

The above mentioned etiological factors permit the entry of the microorganisms responsible for this condition into the perineal region. The poor immune status of the host allows the infection to kick in while the disease spread is facilitated by the virulence of the microorganisms.

UROGENITAL	ANORECTAL	CUTANEOUS
Periurethral infections	Perianal abscess	Occult trauma
Urethral strictures	Complications of:	Complications of :
Bladder carcinoma	Rectal and colonic carcinomas	Vasectomy,
Indwelling catheter	Haemorrhoidectomy	Herniorraphy
Traumatic catheterisation	Rectal biopsy	Superficial skin abscess
Biopsy from prostrate	Sigmoid Diverticulitis and perforation	Fungal infections
	Colo Rectal perforation	
	Crohn's disease	

PATHOPHYSIOLOGY

Fournier's gangrene development requires a source of infection and a portal of entry.The causative pathogens may have low virulence but act synergistically. Necrosis of skin and subcutaneous tissues occurs readily in scrotum and perineal tissues since it lacks subcutaneous tissues [28]. There is no subcutaneous tissue between skin and dartos.

Fournier’s gangrene involves multiple gram (+) and (-) organisms, these organisms are of low to moderate virulence but synergism causes them to progress to severe infections with tissue necrosis and gangrene. It has been believed by many authors that the rapid multiplication and spread of infection in Fournier’s gangrene is due to the polymicrobial nature of the infection which leads to synergistic enzyme production. For example, one organism can cause coagulation of subcutaneous vessels which leads to tissue hypoxia which allows and hastens the growth of facultative anaerobes and microaerophilic organisms. Lechithinase and collagenase are produced by these organisms which lead to rapid spread of the infection due to digestion of fascial barriers.

The pathology includes necrosis of superficial fascial layers and the surrounding tissues: the deep fascia however is not involved and spared.

PATH OF SPREAD IN FOURNIER GANGRENE

THROUGH UROGENITAL ROUTE

- Initially there is infection of paraurethral glands and there is necrosis of skin and subcutaneous layers. The infection is prevented from further spread by the Buck's fascia. Once the Buck's fascia is breached, the infection spreads along the plane of Scarpa's.
- Upward spread thro scarpa fascia is more common than downward spread to perineum through Dartos' and Colle's fascia.

THRO PERINEUM AND RECTAL ROUTE

- Perirectal infections reach near the Sup. Perineal pouch and spread through the Colle's fascia into the testis and scrotum.

FROM THE SKIN

- Trauma and pressure in the perineal tissues due to sitting and tight undergarments result in the spread of bacteria from the skin into the subcutaneous tissue [29].

The pathognomonic pathological features of affected tissue are:

- Fascial layers necrosis.
- Fibrinoid coagulation of the nutrient arterioles

- Infiltration of leukocytes.
- Gram staining and culture may identify and isolate microbes from specimen.

BACTERIOLOGY

Fournier's gangrene is a polymicrobial infection with cultures from the wounds of affected patients showing 2-3 microbial species per case.

Aerobe which was commonly isolated is *E.coli* and anerobe commonly isolated was *Bacteroides spp.*

Frank L. Meleny during World War I reported in China a case series of necrotizing infections and found streptococcal species to be the most common organisms in culture. After his publications Meleney and many others imputed streptococcal species as the sole pathogen for the development of necrotizing infections; however, subsequent clinical series asserted the polymicrobial nature of the all necrotizing infections, including Fournier gangrene.

Currently isolation of a single organism is very rare and there are about 4 different bacterial species commonly isolated along with streptococcus (staph., anaerobic strep, bacteroides and enterobacteriae spp. pseudomonas and

klebsiella) [31]. Lactobacillus gasseri and Candida albicans[32] have also been rarely reported in culture.

Clostridium perfringens, has occasionally been isolated [33]. There have been isolated reports of sarcoptes scabeii and wucherira being isolated.

CLINICAL FEATURES

The typical patient is an adult male in of 50-70 years with characteristic predisposing factors. Females are less commonly affected by this disease.

The hallmark of Fournier's is exquisite **pain and tenderness** in the genitalia, scrotum and perineum. The disease usually progresses through these phases:

1. Malaise and intermittent fever are the initial prodromal symptoms which may last from two days to a week before the appearance of skin changes.

2. Intense pain and tenderness over the scrotum and perineum follows next, along with the appearance of edema of the overlying skin. The intensity of the pain experienced may initially be out of proportion to the findings. It is due to cellulitis and accumulation of fluid.(**COLOUR PLATE 1**)

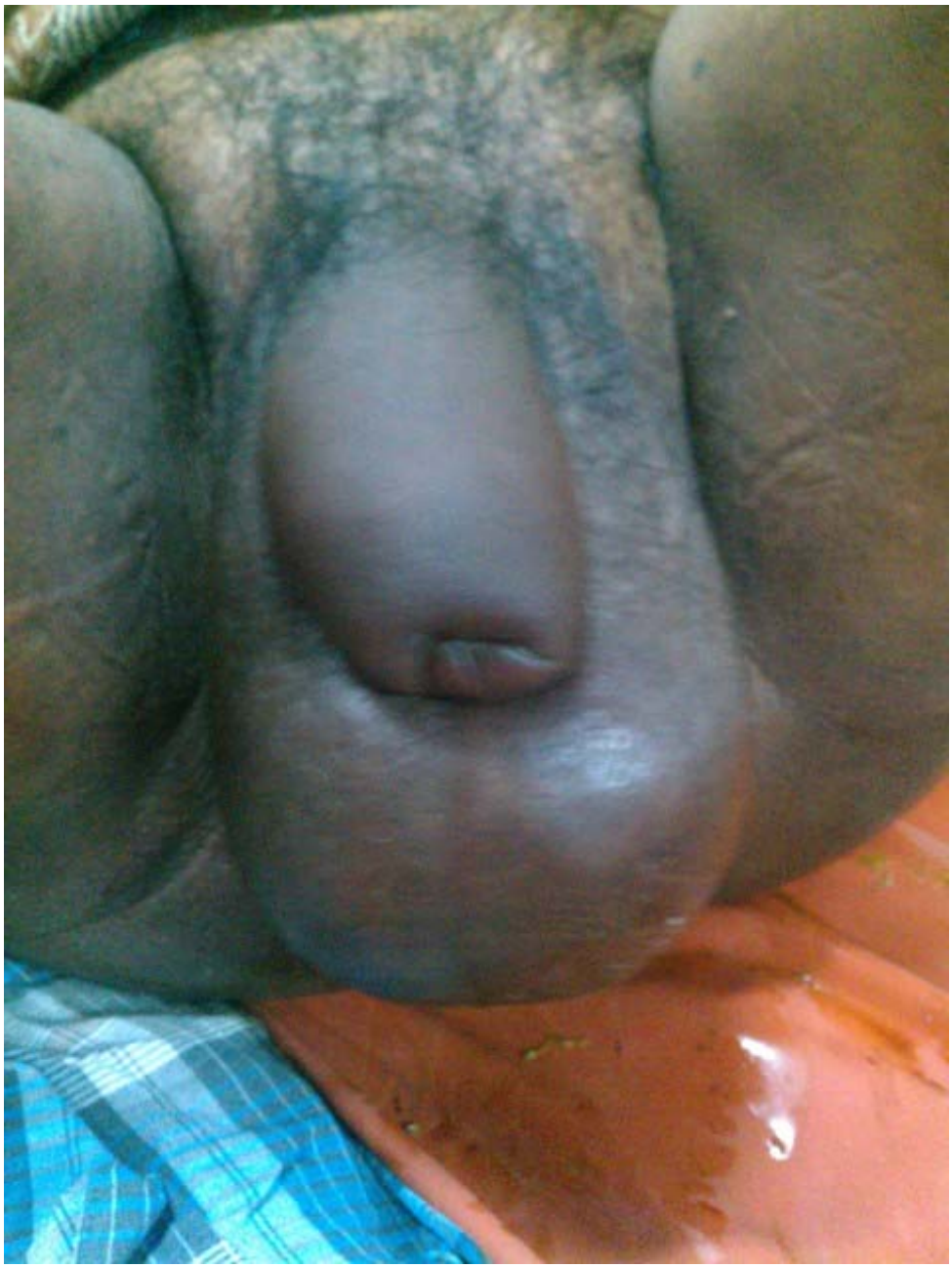
3. Pain and tenderness in local area progressively increases along with erythema of the overlying skin.

4. The overlying skin starts becoming dusky along with appearance of crepitations in the subcutaneous tissue.

5. Lastly Frank gangrene may appear in a region of the perineum and genitalia, Pus starts draining from wounds (**COLOUR PLATE II**).

6. The spectrum of disease ranges from only local symptoms with no sepsis to frank septicemia with SIRS and MODS. It primarily depends on area of involvement and virulence of organism. The host factors are also responsible for this varied spectrum.

FOURNIER GANGRENE INTIAL STAGE.(COLOUR PLATE 1) :



COLOUR PLATE II

PUS DRAINING OUT THROUGH WOUND

INVESTIGATION

Fournier's gangrene diagnosis is essentially clinical and there is no role for additional imaging and blood investigations to confirm the diagnosis and this should not defer and impede the initiation of definitive therapy.

Lab Studies

The lab abnormalities are used to assess the severity and guide to resuscitate the patient preoperatively as most present with severe metabolic disturbances.

Complete Blood Count and Renal Function Test

- For the evaluation of electrolyte derangements.
- Findings suggestive of azotemia. Elevated urea and creatinine
- White cells count to assess the host response to infection.
- Hematocrit may be increased due to dehydration and in late stages of septic shock it may be low.
- Blood sugar levels may be very increased in diabetics causing ketoacidosis .

Liver function test

- Exacerbation of preexisting liver failure.
- Sepsis induced hyperbilirubinemia and hypoalbuminemia.
  - Blood cultures for the identification of any sepsis.
  - For the detection of any sepsis induced coagulopathy, a coagulation profile would serve the purpose (platelet count, fibrinogen level, PT,aPTT and INR)

Others

Any investigation for the assessment of an associated comorbidity and as a part of preoperative anesthetic workup is justified. (eg, chest x ray, ECG, blood group and type)

FOURNIER'S GANGRENE SEVERITY INDEX

In calculating the Fournier gangrene severity indexes score, 9 parameters are measured.

Vital parameters Temperature; Heart Rate; Respiratory Rate;

BLOOD VALUES

Serum Electrolytes namely Sodium, Potassium, and Bicarbonate levels; Hematocrit and white cell count, Creatinine value.

A score was given to each factor based on its variation from normal value. So blood was drawn to measure these parameters. The individual values are summed to obtain the FGSI score.

VARIABLE	SCORE 4	SCORE 3	SCORE2	SCORE 1	0	SCORE 1	SCORE 2	SCORE 3	SCORE 4
Temperature(°C)	>41	39-40.9		38.5-38.9	36-38.4	34-35.9	32-33.9	30-31.9	<29.9
Pulse Rate	>180	140-179	110-139		70-109		55-69	40-54	<39
Respiratory Rate (b/min)	>50	35-49		25-34					<5
Serum Na^+ (mmol/l)	>180	160-179	155-159	150-154	130-149		120-129	111-119	<110
Serum K^+ (mmol/l)	>7	6-6.9		5.5-5.9	3.5-5.4	3-3.4	2.5-2.9		<2.5
Serum Creatinine (mg/100ml)	>3.5	2-3.4	1.5-1.9		0.6-1.4		<0.6		
Haematocrit (%)	>60		50-59.9	46-49.9	30-45.9		20-29.9		<20
WBC (Total/mm^3 x 1000)	>40		20-39.9	15-19.9	3-14.9		1-2.9		<1
Serum Bicarbonate (mmol/l)	>52	41-51.9		32-40.9	22-31.9		18-21.9	15-17.9	<15

IMAGING IN FOURNIER GANGRENE

X RAY OF LOCOREGIONAL AREA

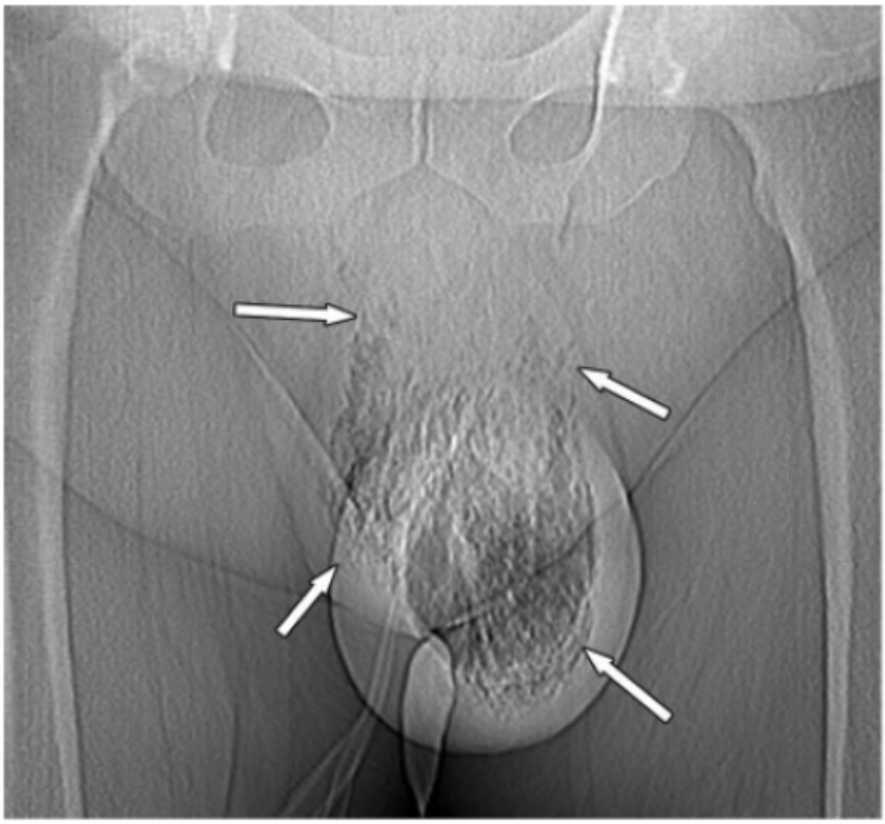
Conventional radiography is indicated when the findings by physical examination are not definitive. Radiography is more reliable for the detection of gas within the soft tissues.

Surgical exploration is definitely indicted when there is subcutaneous crepitus or gas in the soft tissues.

Gas in the soft tissues over the scrotal and perineal regions may be seen in X-rays as hyperlucencies. Inguinal regions, thighs and anterior abdominal walls are other regions also affected by subcutaneous emphysema that has spread from the scrotal and perineal regions (**COLOUR PLATE III**).

However, upto 10% of patients finally diagnosed with Founier's gangrene did not have air in the subcutaneous tissues overlying the perineal and scrotal regions[35].

COLOUR PLATE III



ULTRASONOGRAM OF ABDOMEN, SCROTUM AND PERINEUM

- A thickened scrotal wall with hyperechoic foci due to gas, is seen on ultrasonography as “dirty” shadowing(**COLOUR PLATE IV**).

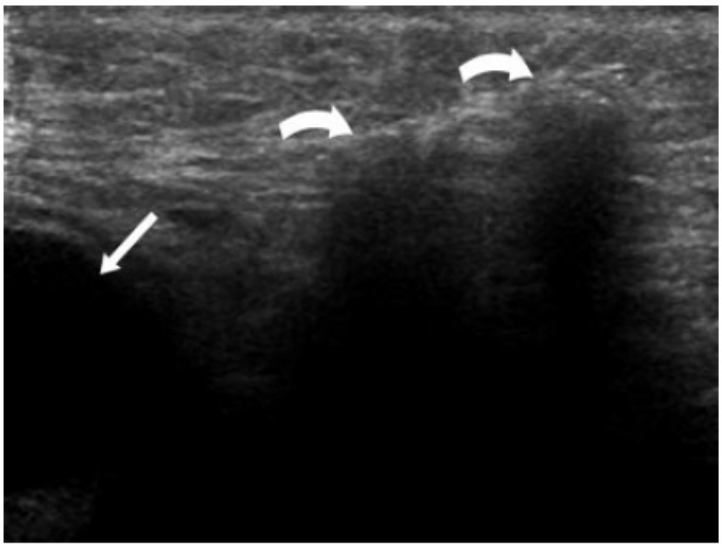
- Clinical evidence of crepitus is usually preceded by radiographic evidence of gas in the scrotal wall. There is also a possibility of occurrence of hydroceles unilaterally or bilaterally.

- An intra-abdominal or retro-peritoneal source of infection may be responsible for testicular involvement which can be identified in ultrasound.

- Inguinoscrotal incarcerated hernia may be differentiated from Fournier’s gangrene by ultrasonography by the presence of gas in the obstructed bowel lumen, rather than the gas in scrotal wall.

- If a diagnosis of testicular torsion is being considered, Ultrasonography may be useful in assessing the testicular blood flow.

- **COLOUR PLATE IV**
- **ULTRA SOUND OF SCROTUM**



- **ARROW SHOWING SCROTAL WALL EDEMA**
- **CURVED ARROW SHOWING HYPERECHOIC AREAS**

CT scanning

CT will prove to be useful, in patients in whom the diagnosis is in doubt or the disease extent cannot be made out clearly clinically with certainty. For the assessment of disease extent, CT is more specific than ultrasound or Xrays.

Progressive infiltration of soft tissues can be seen on CT in early stages of Fournier's gangrene even before the appearance of subcutaneous emphysema.

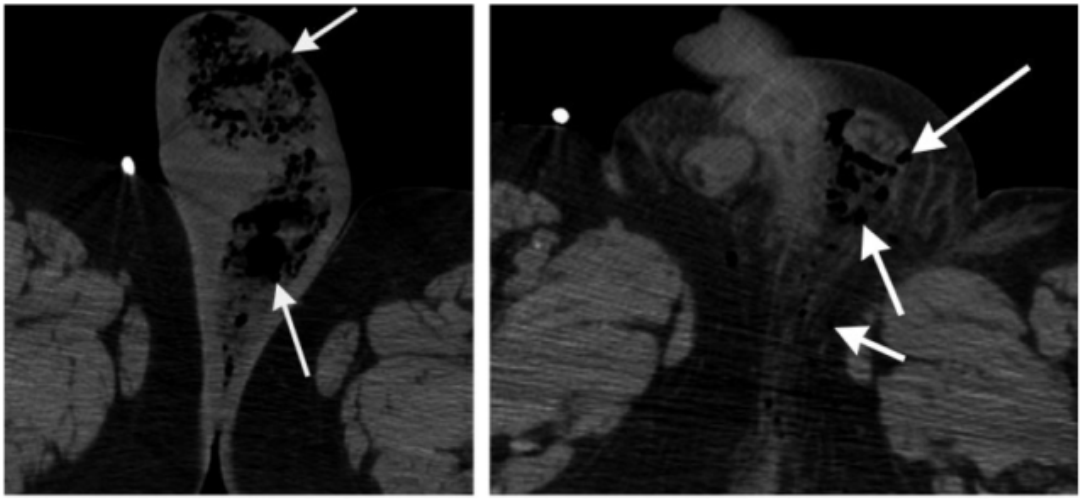
Findings suggestive of Fournier's gangrene on CT are:

- Thickening & inflammation of soft tissues.
- Fluid collection/abscess
- Stranding of fat around affected structures
- Asymmetrical fascial thickening
- Subcutaneous emphysema caused by gas forming bacteria(**COLOUR PLATE V**)

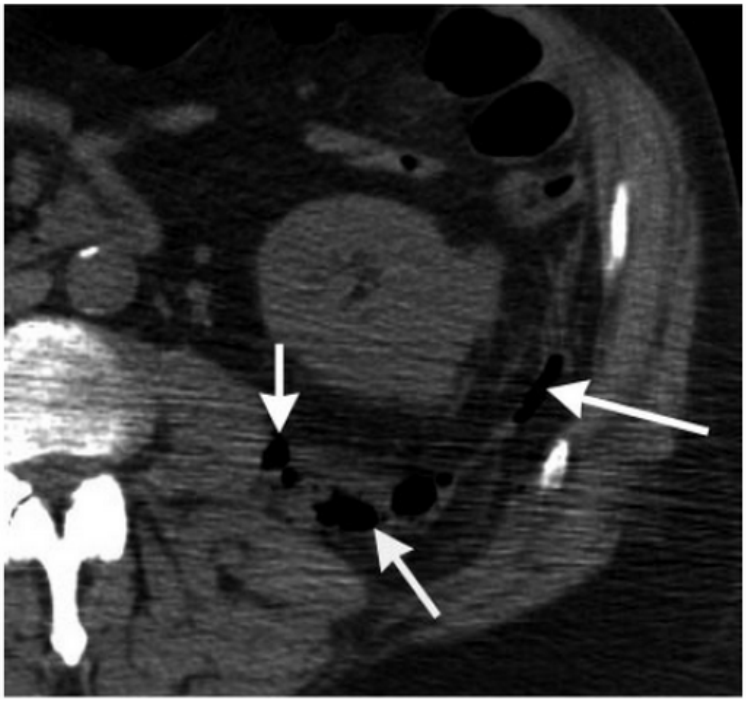
In this condition, the subcutaneous emphysema spreads along fascial planes, reaching as far out as the anterior abdominal wall, thighs, retroperitoneum and thoracic wall which can visualized with CT scanning. (**COLOUR PLATE VI**)

COLOUR PLATE V

CT SHOWING GAS IN SCROTUM EXTENDING TO INGUINAL REGION



COLOUR PLATE VI



CT SHOWING EXTENSION TO PARARENAL SPACES

TREATMENT

Fournier's gangrene is a surgical emergency. The therapy involves preoperative hydration and Hemodynamic stabilization , administration of broad spectrum antibiotic and urgent and immediate surgical debridement of necrosed area .

The most important aspect of management is urgent Surgical Debridement since the necrosis spreads very rapidly. The rate of spread of infection can be as high as two centimeters in one hour[37].

The antibiotic administration should start immediately on admission which should cover and be effective against staph. strepto and coliforms. A separate antibiotic administration should be added to cover anaerobes.

Urgent resuscitation with fluids as well as blood transfusions may be needed.

The disease left in its course of events leads to regeneration of scrotum skin and epitheliazation and scar formation [38].There are reports of complete healing and skin closure without any need for further surgeries [39,40].

PHARMACOTHERAPY FOR HEMODYNAMIC OPTIMIZATION

The two motives of the medical treatment are restoration of organ dysfunctions and treatment of the infection.

Most of our patients present late to the hospital, hence they are dehydrated and present with features of systemic sepsis. There is an absolute need for urgent restoration of organ perfusion and correction of dehydration and electrolyte disturbances before the surgery.

Ionotropic support may be needed in cases of hypotension. Nor adrenaline is needed in cases of septic shock.

Treatment also involves the institution of broad-spectrum antibiotic therapy. Providing coverage for grampositive, gram-negative, aerobic, and anaerobic bacteria is essential. The antibiotic spectrum should cover staphylococci, streptococci, the Enterobacteriaceae family of organisms, and anaerobes.

Penicillin group of drugs like Ampicillin is used to cover streptococci while metronidazole is given against anaerobes such as bacteroides. A broad spectrum antimicrobial agent, preferably the third generation Cephalosporins combined with Aminoglycoside amikacin or gentamicin is used against gram negative organisms such as the coliforms.

Clindamycin may be used as it is shown to suppress toxin production and modulate cytokine production. Linezolide and daptomycine, tigecycline is

warranted in cases of previous hospitalizations with prolonged antibiotic therapy which may lead to resistant bacteroides.

New clinical guidelines currently recommend the use of Carbapenems (Imipenem, meropenem, ertapenem) or piperaziline-tazobactam. These newer drugs have larger distribution and lesser renal toxicity in comparison to aminoglycosides. A relook at the classical triple therapy should be done and this should be replaced by newer drugs as and when required.[40].

If fungi show up on initial tissue stains like potassium hydroxide stain or it is grown in culture an antifungal like caspofungin or amphotericin B should be added.

Intravenous immunoglobulin therapy has been found to be a good adjunct in sepsis following proper surgical debridement and good antibiotic cover and it is found to hasten the recovery.

SURGICAL MANAGEMENT

- **DIAGNOSIS IN DOUBTFUL CASES**

- In patients clinically when there is a suspicion of fournier , a definitive diagnosis is arrived at by incising and examining the most suspicious area under anaesthesia.

- Sometimes before the appearance of gangrene , the disease presents as severe cellulitis. In such cases, the fascia appears edematous rather than the typical gray-black appearance on surgical incision and removal of overlying skin.
- On finding Frank gangrene and/or purulent material in subcutaneous tissue it establishes the diagnosis.

- **RADICAL REMOVAL OF NECROTIC AREAS (COLOUR PLATE VII & VIII)**

o Skin may be deroofed or multiple incisions may be made to lay open the subcutaneous layer. This allows the exudates to come out also allows us to inspect the progress of disease.

o Excise and remove completely all necrotic non-viable tissue until viable well-perfused tissue is visualized

COLOUR PLATE VII

INTIAL PRESENTATION

POST DEBRIDEMENT SPC DONE FOR URINARY DIVERSION

COLOUR PLATE VIII
AT OPD FOURNIER GANGRENE PATIENT

POST DEBRIDEMENT (Patient catheterized for diversion)

PICTURE AFTER 15 DAYS

The cutaneous involvement underestimates the actual spread of the disease as it is less than the subcutaneous tissue involvement.

- Tissue should be sent for hispathological examination and culture for anaerobic and aerobic organisms.

- The extent of the disease and necrosis should be reassessed as the disease may progress and also intial debridement may not be complete due to poor general condition of the patient.

- Repetitive operative debridement at a later date may be necessary to ensure removal of infective focus and gangrenous areas.

- Antibiotics are guided based on wound and tissue culture and sensitivity. It must be continued for 10-14 days or till healthy granulation appears.

REQUIREMENT OF ORCHIDECTOMY

- Involvement of the testes is a documented complication in Fournier's gangrene, in spite of the fact that the testes have their own independent blood supply. The need for orchidectomy arises when the infective process spreads to the retroperitoneal from the intra abdominal compartment. Retroperitoneal spread foretells poor prognosis.

- Upto 21% of patients in some series have required orchidectomy surgery for non-viable testes.

DIVERSION PROCEUDRES

In cases where there is widespread perineal involvement and constant fecal soiling, a colostomy may be needed to reduce wound contamination by faeces.

Fecal diversion

- In patients with severe perineal involvement, colostomy has been resorted for fecal diversion. The logic behind this approach is the involvement of the anal sphincter by this condition leading on to fecal incontinence and contamination of the wound margins by feces.
- Hence when a rectal diversion is done, it would facilitate better wound healing by causing a net reduction in the number of microorganisms infecting the perineal region. (**COLOUR PLATE IX**)
- **COLOUR PLATE IX**

Case requiring colostomy

Rectal diversion devices

- The Flexi-seal Fecal Management system functions in the same way as a colostomy to prevent fecal contamination of wounds. It is a catheter made of silicone which serves for fecal diversion in patients with diarrhea, local burns or skin ulcers.
- Estrada et al found it to be an effective alternative to colostomy. The anal canal should be examined prior to catheter placement. It also offers advantages over stomas in terms of cost effectiveness and recovery of the patient psychologically.
- \\ The presence of stricture or stenosing lesion or presence of carcinoma precludes the use of this device.

Urinary diversion.

- Penile and urethral involvement necessitates urinary diversion. Urinary catheterization is sufficient in some cases. Supra pubic cystotomy needs to be done in cases with stricture urethra and cases with BPH (**COLOUR PLATE X**).

COLOUR PLATE X STRICTURE URETHRA WITH SPC

Topical therapy

- When unprocessed natural honey was applied over debrided areas, it produced good results within a week of application by virtue of its low pH (3.6) and necrotic tissue digesting enzymes.
- Post operative application of hydrogen peroxide and use of 0.5% sodium hypochlorite have been advocated.
- Application of lyophilized collagenase as a form of enzymatic debridement has also produced favourble results.

Local negative pressure treatment (VAC)

The wound VAC device is a form of occlusive dressing that is applied around a sponge placed immediately over the wound. Through an opening in the occlusive dressing suction is applied through tubing connected to a negative pressure device. It needs to be shaped so as to fit into irregular wounds and cavities. It is advisable to change it once in 2-3 days. It serves to promote granulation tissue formation, improves local blood flow and brings down the oedema. Going by the number of reports, VAC seems to be increasingly used for patients with Fournier's gangrene[47,48].

Advantages stated are:

(a) There is decreased need for repeated debridements.

(b) One time reconstruction can be accomplished because of the reduction in raw area and appearance of healthy bed for better graft or flap survival.

VAC also has the advantage of promoting the uptake of skin graft by holding the graft in situ and preventing the accumulation of blood or fluid beneath it.

Disadvantages associated with this device are the need to immobilize the patient and high cost.

Hyperbaric oxygen therapy

- This is a method of improving oxygenation to the arterial and tissues by making the patient breathe 100% oxygen while he is put in an environment of high ambient pressure.
- It inhibits growth of anaerobic organisms, promotes fibroblast proliferation and angiogenesis, facilitates intracellular transport of

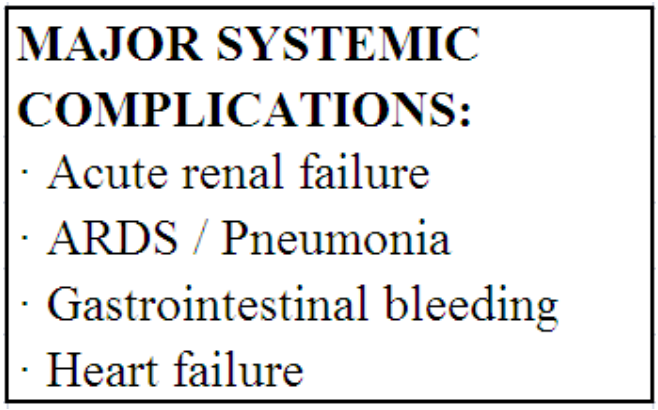
antibiotics, optimal neutrophil phagocytic function and reduces edema by vasoconstriction [43] .

- Hyperoxia promotes transmembrane transport of aminoglycosides through an oxygen dependent pump increasing its efficacy.
- Possible side effects reported are barotrauma to middle ear and CNS toxic effects.
- Hyperbaric Oxygen Therapy continues to be a matter of debate till date.

Nutritional support

- Stress in septicemia patients causes a phenomenal increase in basic energy requirements. Multiple surgical procedures and mechanical ventilation rules out oral intake as a possible source of energy.
- In this situation, total parenteral nutrition alone may not ensure adequate calorie intake. Studies have revealed that critically ill patients have calorie requirement of about 125% of the basal quantity. This can be ensured by additional enteral intake.
- The theoretical benefit of the intake of oligoelements (like arginine, citrulline and glutamine) has been discussed in many articles.
- Low plasma concentrations of L-arginine, documented in these patients has been correlated with poor outcomes. This is because ariginine promotes wound healing and improves the immune function.

COMPLICATIONS



Renal function

- Due to septicemia and release of toxins to the blood stream there is renal failure in most patients. Most cases are managed by fluid management and other conservative measures.

- Some cases require hemodialysis to manage the acute renal failure.

- Intravenous albumin is found to improve the survival and also hasten the recovery in cases of septicemia

There may be **UNRELENTING SEPSIS** despite surgical debridement which may be due to the following factors:

- Incomplete removal of infective process causing progress and ascend of necrosis.

- There may be other focus of infection which is not identified initially, for example colonic diverticular abscess or hollow viscus perforation.
- Sepsis induced Acute respiratory distress syndrome.
- Pnuemonia and lung collapse due to chronic bed confinement.
- Deep vein thrombosis and pulmonary embolism due to prolonged immoblisation and associated risk factors like carcinoma.

Causes of mortality are given below:

Causes of death
Sepsis induced MODS
Acute renal failure
DIC
Diabetic ketoacidosis

MORBIDITY DUE TO FOURNIERS

Morbidity due to Fournier's disease varies corresponding to the hospital stay duration which ranges from 2 days to a reported maximum till around 280 days [(44)].

Although spontaneous healing with simple dressing has been reported, others advocate avoiding it, as there may fixity of testis to perineum and surrounding structures and immobilization could result by it.

OUTCOME AND PROGNOSTIC FACTORS

Mortality rates: 3 - 30% **Poor prognostic factors**
Advanced Age
Anorectal causes
Number of organ failure at admission
Delay in presentation & treatment
Diabetes

Extent of involvement, associated comorbidities,pre hospital symptom duration are important prognostic factors. Any undue delays in diagnosis and initiation of treatment also determine the final outcome. The severity index score has a definitive role in predicting the mortality and morbidity.

The death rate in Fournier's gangrene is lesser than that is associated with other forms of necrotizing fasciitis and is probably due to the gravity dependent drainage that occurs in perineum and scrotum. The mortality rate in Foumier's gangrene varies from 3 to 30 per cent.

FOURNIER'S GANGRENE SEVERITY INDEX

There are many independent prognostic factors described to predict the mortality for Fournier gangrene namely, Diabetes, advanced age, primary anorectal source of infection, delayed presentation, systemic sepsis on admission.

Laor et al. in 1995 and designed the FGSIS (Fourniers's Gangrene Severity Index Score) which is a modification of the already known APACHE II (Acute Physiology and Chronic Health Evaluation II) in which they found that patients with a score around 7 survived and score greater than nine had significant mortality .

The score has an advantage that it can be very easily calculated based on vital parameters and basic blood parameters. No sophisticated investigations and cost is involved in it. Lower cost factor offers advantage to this group of patients because the disease commonly affects lower socioeconomic status.

Laor et al reported that a score greater than nine had mortality as the outcome with positive predictive value of 80%. The score below nine had probability that they survived. This cutoff point has been achieved through ROC curve in many other studies. In many subsequent studies the value of score which best predicted mortality was nine. So a score above nine should

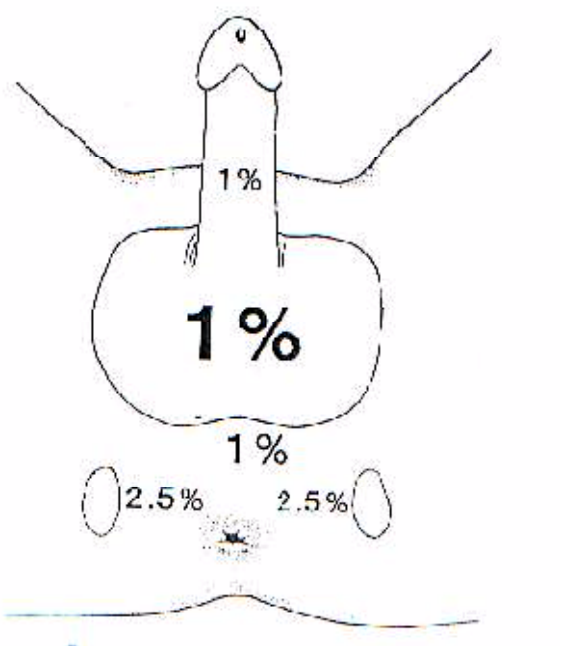
alert the treating surgeon to pursue aggressive resuscitative measures and radical surgical drainage.

Specific metabolic parameters (serum albumin and alkaline phosphatase), predisposing factors and disease extent should be assessed together to predict treatment outcome and survival.

It has been proved that survived patients had significantly higher serum albumin consequent to better patient's metabolic reserve.

Noteworthy, FG has predilection towards immune dysfunction, and patients who had a higher IL-2 level had more survival [(45).] Interleukin is proved to play a role in augmenting the host response and hence restricting the infection. In a study the IL-2 value was added to FGSI score and a cutoff for mortality was detected[(46)].

Abdul Samad in 2007 devised a new system to predict mortality in which the extent of involvement and platelet count value was calculated and added to the already mentioned FGSI score **(COLOUR PLATE XI)**



He proved in his study that score on admission > 12 has high chance of mortality. Since the sample size was small there was not any statistical significance to the result. It needs to be reevaluated at a large scale study to prove its usefulness and to accept it in future.

COLOUR PLATE XI

FOURNIER GANGRENE IN PLHA PATIENT WITH EXTENSIVE INVOLVEMENT OF THIGH AND PERINEUM

RECONSTRUCTION

Wound cover is achieved by following means

- **Healing and epithelisation without grafting(COLOUR PLATE XII)**
- **Secondary skin closure.**
- **SSG to the raw area.**
- **Testis implantation into thigh**
- **Orchidectomy and skin closure(In elderly)**
- **Medial thigh flap**

RECONSTRUCTION

- Placement of bare testes in the medial part of thigh in subcutaneous plane. It has higher success rate , short operative time and needs no much surgical skill. Young individuals may find it unacceptable , there is chance that higher temperature in thigh may affect fertility. **(COLOUR PLATE XIII).**
- In consenting older patients the option of orchidectomy and skin closure can be done. But patient may be psychologically affected due to loss of testis and affect his sexual function **(COLOUR PLATE XIV).**

- Split thickness skin graft can be put over exposed testis but the disadvantage is the movement of testes is lost and it gives a sticky feel when the raw surface is adhered to the graft.

- Tight undergarments should be avoided because it may cause constant friction of graft and then make it prone to infection.

- Rotation flap from thigh can be used to cover the testis. cutaneous supply to the skin is maintained so it is sensitive to touch sensation. Patients have a good space enabling free movement of testes **(COLOUR PLATE XV)**.

COLOUR PLATE XII

Healing and epithelisation without grafting

COLOUR PLATE XIII

BARE TETSTES POST DEBRIDEMENT

MEDIAL THIGH POUCH PLACEMENT

COLOUR PLATE XIV

AFTER ORCHIDECTOMY PRIOR TO CLOSURE

COLOUR PLATE XV

HARVESTING A THIGH FLAP

OBSERVATION AND RESULTS

The study was conducted in surgical wards of Govt. Stanley Medical college hospital. The following are my observations regarding the epidemiology and etiological , clinical features and treatment of patients affected with Fournier's gangrene.

AGE AND SEX

The following table (TABLE 1) gives the age distribution of the patients.

TABLE I

AGE GROUP	SURVIVORS (n=29)	DECEASED(n=6)	% GROUP
40-45	4	NIL	11.4
45-50	6	NIL	17.1
50-55	9	NIL	25.7
55-60	2	1	8.5
60-65	2	3	14.2
65-70	3	1	11.4
70-75	2	0	5.7
75-80	1	1	5.7

The average age of patients in this study was **56.5 ± 10.2 years**.

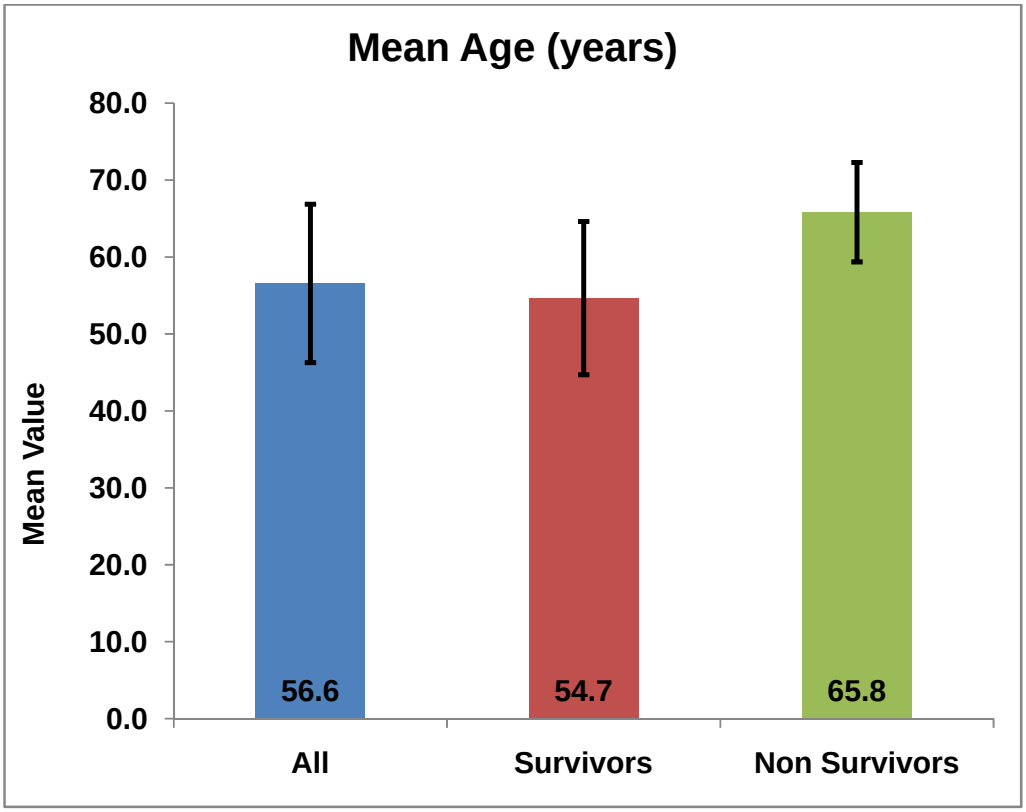
Mean age of all patients

	N	Mean	Std. Dev
AGE	35	56.57	10.296

The mean age **of survivors** was **54.6** and mean age of **deceased** patients was **65.8** years. (**GRAPH I**)

In the study 34 patients were male and we had **one was female patient**.(**GRAPH I**)

GRAPH I



PREDISPOSING FACTORS

The risk factors found in these patients were diabetes, chronic renal failure and immunosuppression.

- 30 patients were found to be diabetic and it is the most common underlying disorder in this group of patients i.e. 85% of the subjects.

- Pre existing chronic renal failure was found in 4 patients and 2 patients had HIV sero-positivity.

- Alcoholism was common denominator in most of the patients.

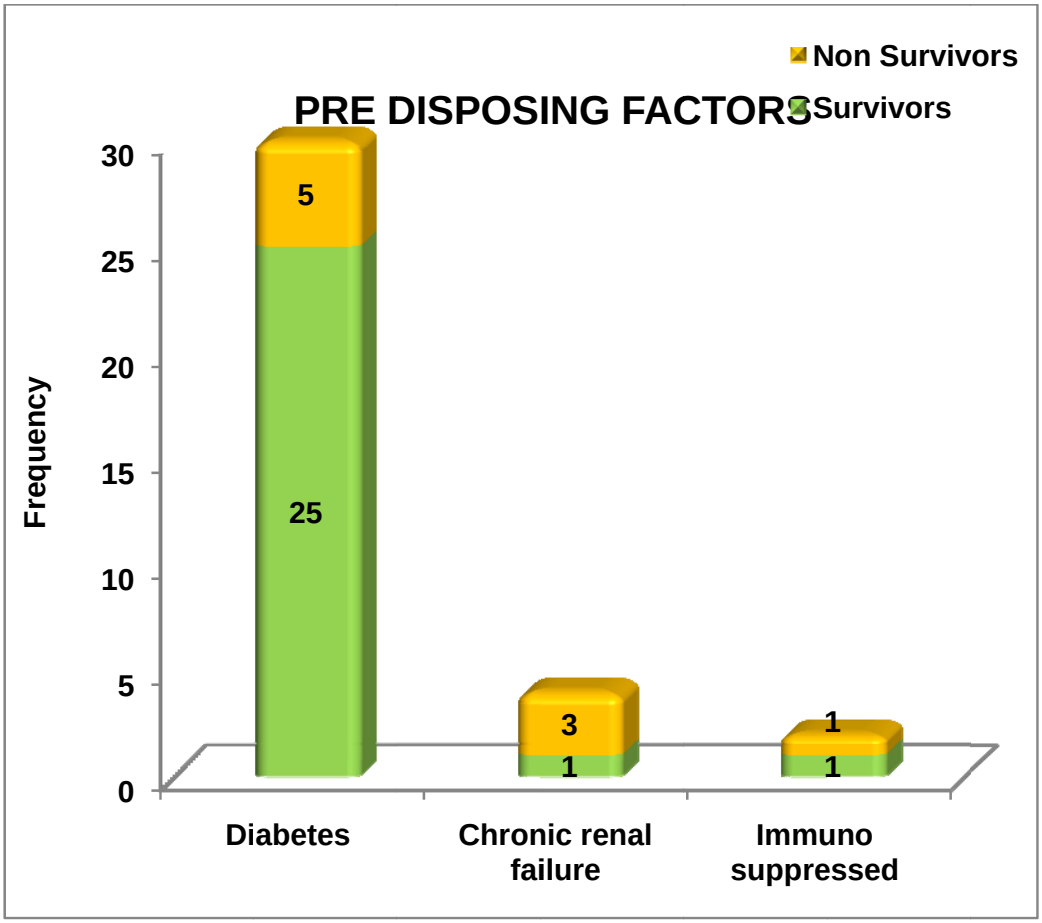
- 2 out of 35 patients had no pre existing disease.

- 5 out of 6 patients who died had diabetes.

TABLE II

PRE DISPOSING FACTORS	TOTAL	SURVIVORS(n=29)	NON SURVIVORS(n=6)	% OF TOTAL
DIABETES	30	25	5	85.7
CHRONIC RENAL FAILURE	4	1	3	11.4
IMMUNO SUPPRESSED(HIV/CANCER)	2	1	1	5.7
NONE	2	2	0	5.7
ALCOHOLISM				

GRAPH II



ETIOLOGY

A recognizable etiological factor was found in 65 % of cases i.e.23 cases. 12 cases had no etiological source.

The most common source of infection was from the colorectal area. 15 cases had colorectal etiology of which 10 were due to peri anal abscess.2 cases had fistula in ano as the previous etiological factor.

Rectal carcinoma was found in 2 cases. 1 case was already receiving chemo radiation for the cancer. Another had received chemo radiation before 1 year and lost follow up after that.

In 1 case there was post radiation induced tinea cruris which was the etiological factor. In patients with HIV(2 cases), cutaneous infection was instrumental in initiation and propagation of infection.

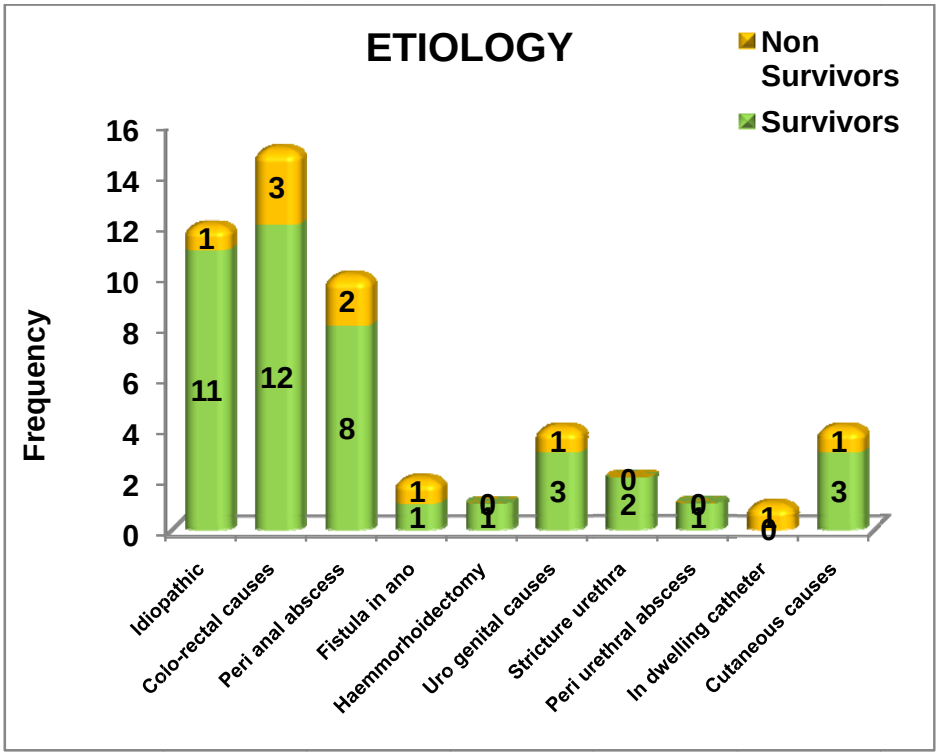
1 case had indwelling catheter for past 6 months (for BPH) following which he developed Fournier's gangrene.

Among the deceased the most common causes was from colorectal sources.

TABLE III

ETIOLOGY	NO. OF PATIENTS	PERCENTAGE	SURVIVORS	DECEASED
IDIOPATHIC	**12**	**34.3**	11	**1**
COLO-RECTAL CAUSES	**15**	**42.8**	12	**3**
PERI ANAL ABSCESS	10		8	2
FISTULA IN ANO	2		1	1
CARCINOMA	2			
HAEMMORHOIDECTOMY	1		1	0
URO GENITAL CAUSES	**4**	**11.4**	3	**1**
STRICTURE URETHRA	2		2	0
PERI URETHRAL ABSCESS	1		1	0
IN DWELLING CATHETER	1		0	1
CUTANEOUS CAUSES	**4**	**11.4**	3	**1**

GRAPH III



DURATION OF SYMPTOMS

The symptom onset is defined as the appearance of gangrenous changes, purulent discharge in skin, and elicitation of fluctuation. Most of the patients in this study presented late after the development of symptoms. Mean duration of symptoms in all the subjects was 2.99 days with a standard deviation of 1.13 days

TABLE IV

	N	**Mean**	**Std. Dev**
DURATION OF SYMPTOMS(DAYS)	35	2.99	1.128

The mean duration of symptoms among the survivors was 2.66 days and mean duration of symptoms among deceased was 4.58 days.

Mean duration among survivors and deceased with standard deviation(**TABLE V**).

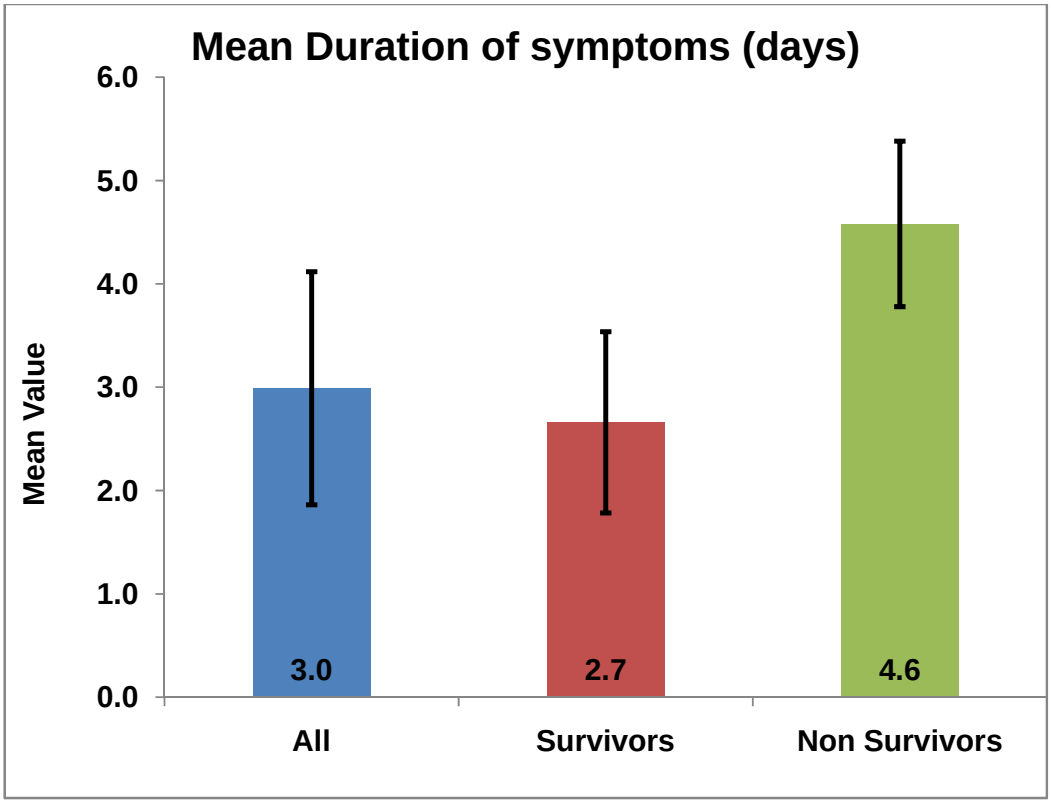
TABLE V

Variable	**MORTALITY**	**N**	**Mean**	**Std. Dev**	**P-Value**

DURATION OF SYMPTOMS (DAYS)	No	29	2.66	0.877	<0.001
	Yes	6	4.5	0.801	

Late presentation i.e. increased duration of symptoms before presentation to hospital was associated with increased mortality and this was found to be statistically significant.

GRAPH IV



CLINICAL FEATURES

In all patients there was history of preceding symptoms of pyrexia, myalgia and lethargy which varied between 2-5days.

They also had history of pain in the local area throbbing in nature due to collection of pus . This was followed by obvious gangrene of portion of genitalia and turbid discharge and sloughing out of skin with exposure of underlying structures.

Local gangrene without features of systemic sepsis was found only in 14 patients. 21 patients had signs of sepsis as evidenced by tachycardia, fever, respiratory distress and renal failure . Sepsis was more noted in elderly patients, those with delayed consultation and those with co morbid disease like diabetes, alcoholics etc,.

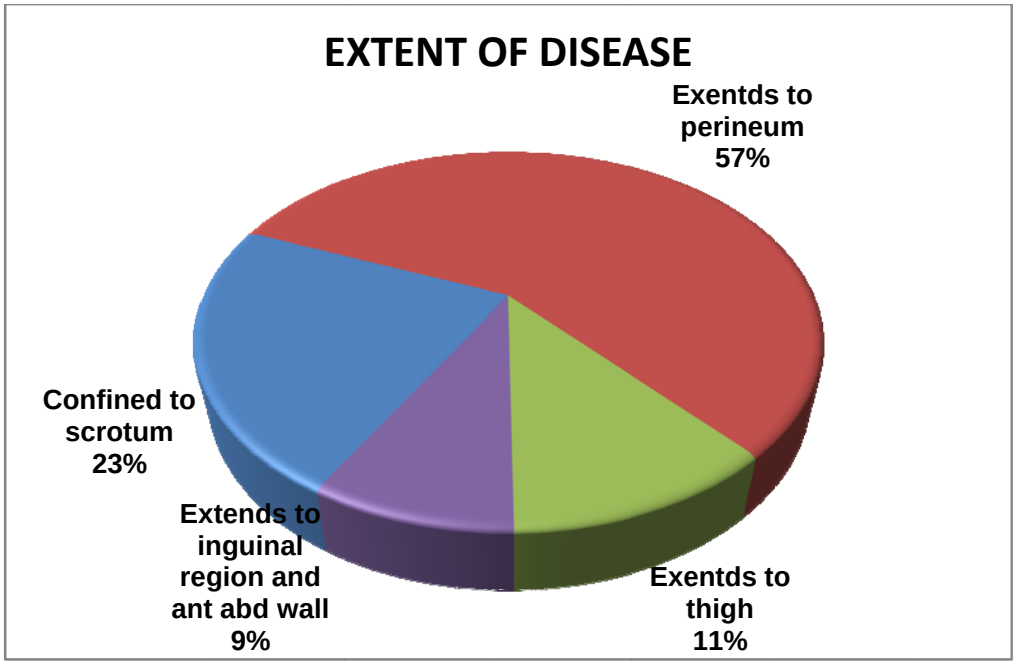
The extent of gangrene varied in each patient(TABLE VI). Being a spreading infective process, the gangrene was either involving the part or whole of scrotum and some with extension to the penis, perineum, thigh and anterior abdominal wall.

In this study, gangrene was confined to scrotum in only 8 patients, with extension into perineum in 20 patients. 4 patients had spreading extensive gangrene to the perineum, medial aspect of thigh. 3 cases had extension to anterior abdominal wall.

TABLE VI

EXTENT OF DISEASE	NO. OF PATIENTS(N=35)	PERCENTAGE
CONFINED TO SCROTUM	8	5.7
EXENTDS TO PERINEUM	20	57.1
EXENTDS TO THIGH	4	11.4
EXTENDS TO INGUINAL REGION AND ANT ABD WALL	3	8.6

GRAPH V



LAB STUDIES

Laboratory investigations were done to assess the extent of systemic toxicity. We looked for electrolyte disturbances, glucose levels, and presence of ketosis, urea creatinine values to assess organ dysfunction.

Approximately 72% of patients had raised urea, creatinine values, suggestive of pre renal azotemia, on the day of admission. 26 out of 35 patients had Acute Kidney injury on admission. Six patients needed hemodialysis. Rest of the patients the renal dysfunction resolved with fluid management and treatment of sepsis namely surgical debridement and antibiotic therapy.

Diabetic ketoacidosis was present in 8 cases. Uncontrolled DKA due to sepsis was the cause of death in one patient.

Wound swab and pus culture and sensitivity were sent for all patients on admission and thereafter during hospital stay at 5 days interval.

The organisms isolated were Streptococci, E.coli, Pseudomonas, and Klebsiella and cogulase negative Staph. aureus.

TREATMENT

The mainstay of treatment of patients with fournier gangrene is pre operative resusicitation, surgical debridement and antibiotic therapy.

Debridement resulted in exposure of testicles in patients with extensive involvement of scrotum.

Regular cleaning and dressing with hydrogen peroxide and povidone iodine solution was done for all patients, until healthy granulation appeared.

In 2 patients, Orchidectomy was done. In one case it was due to involvement of testes due to spread of disease to retroperitoneum. In other case orchidectomy and the skin closure was done during reconstruction.

Colostomy for fecal diversion was needed in two cases. In both cases reversal was done after three months.

Suprapubic cystostomy for urinary drainage and diversion was done in 3 cases. Three had stricture urethra and had extensive penis and perineal involvement where catheterization was not possible.

Out of 35 patients the following were the reconstructive procedures adopted.

TABLE VII

Healing and epithelisation without grafting	6	20%
Secondary skin closure	13	44%
Skin grafting	5	17%
Testes placement in Medial thigh	3	10%
Flap cover	2	6%

In patients with exposed testicle, medial thigh implantation of testis was done in 3 cases and in 2 patients medial thigh flap was done a reconstructive procedure.

COMPLICATIONS

- The common complications were Acute kidney injury and septicemia.
- 26 out of 35 patients had evidence of pre renal azotemia on admission. About 6 patients needed hemodialysis.
- All patients (6/35) who died had acute renal failure and there were two patients among them who underwent Hemodialysis.
- Systemic Sepsis was seen in 21 patients. It was immediate cause of death in one patient.

FOURNIER'S GANGRENE SEVERITY INDEX SCORE

The FGSI score was calculated based on clinical examination and lab values.The mean score of all patients in this study was 5.49.

TABLE VIII

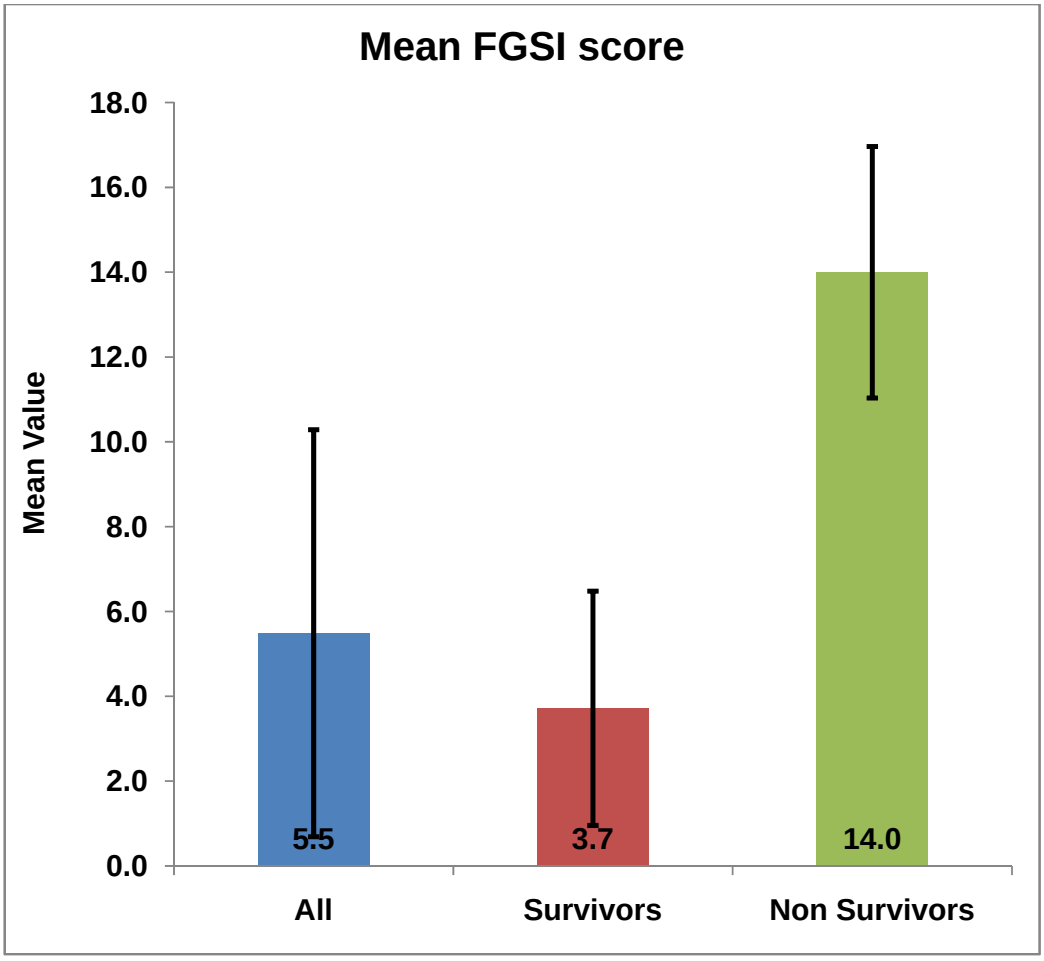
	N	Mean	Std. Dev
FGSI SCORE	35	**5.49**	4.798

The mean score of survivors were 3.72 and the mean score of deceased where 14. This score predicting mortality was found to be statistically significant. Mean score among survivors and deceased.

TABLE IX

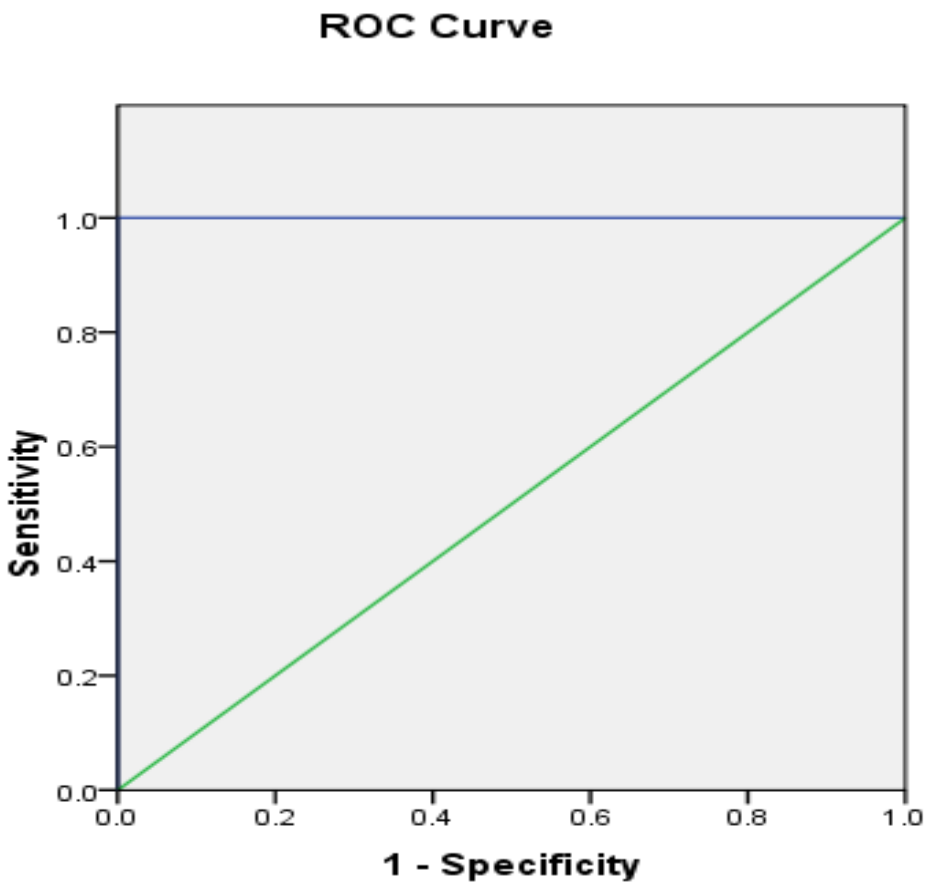
	MORTALITY	N	Mean	Std. Dev	P-Value
FGSI SCORE	No	29	3.72	2.763	<0.001
	Yes	6	14.00	2.966	

GRAPH VI


Mean FGSI score
Mean Value
18.0
16.0
14.0
12.0
10.0
8.0
6.0
4.0
2.0
0.0
5.5
3.7
14.0
All
Survivors
Non Survivors

ROC CURVE analysis was done find out the best cut off point of FGSI SCORE which predicts the mortality.

GRAPH VII



Area under the Curve = 1.000

The ROC curve analysis predicted that the FGSI score of 11 or more will predict the non-survival status.

TABLE X

FGSI SCORE		MORTALITY		Total
		Yes	No	
	≥ 11	6	0	6
	<11	0	29	29
Total		6	29	35

TABLE XI

	Estimate	Lower - Upper 95% CIs
Sensitivity	100%	61.0, 100.0
Specificity	100%	88.3, 100.0
Positive Predictive Value	100%	61.0, 100.0
Negative Predictive Value	100%	88.3, 100.0
Diagnostic Accuracy	100%	90.1, 100.0

A FGSI score of 11 on admission best predicted the mortality in our study.

DISCUSSION

The age group defined initially by Fournier was young male. In 1940 the average age group affected was found to be around 40 years. . However in recent case series and large scale studies it was found that pre disposed ages was from 50-70 years. The average age in our study was found out to be 56 years.

The reason why old age is predisposed is they possess all risk factors required for the development of disease.

Age was described as a independent risk factor in the literature by Kamer et al, Bosglu et al. It was observed in their studies that subjects with **Age > 60** years had increased chances of mortality.

The **mean age of deceased** in our study was **65.8 years (p value 0.013) (TABLE XI)**.

TABLE XI

	OUTCOME	**N**	**Mean**	**Std. Dev**	**P-Value**
AGE	**SURVIVORS**	29	54.66	9.954	0.013
	DECEASED	6	65.83	6.463	

The male female ratio in this disease in most series was found to be 10:1. There are many isolated case reports of fournier's gangrene in female. But it was eke who formally calculated the sex ratio in a metaanalysis of over thousand seven hundred cases.

There was one female case among 35 patients. She had initially a vulval abscess (cutaneous source of infection) which progressed to Fournier's.

PRE DISPOSING FACTORS

The most common underlying disorder is diabetes and the first reported case in 1700 s was probably a diabetic. Diabetes was found in about 85% of our cases. In most studies there were about 75-80% of patients affected with diabetes.

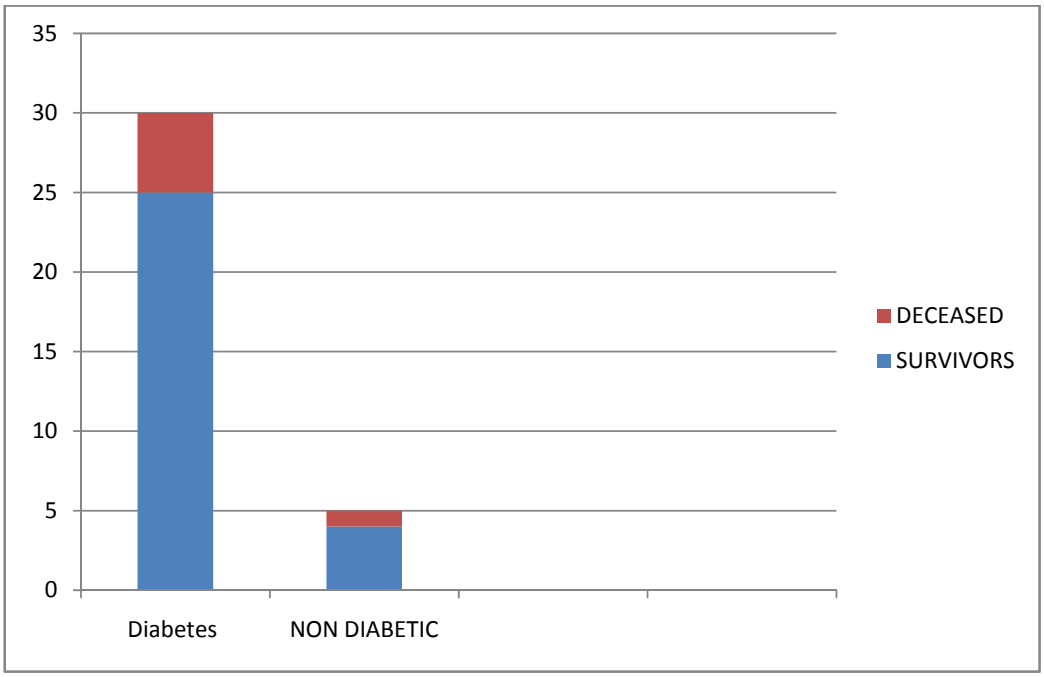
Korkut , Unalp and Eke defined diabetes as a independent risk factor.

5 out of 6 patients who died had diabetes. Diabetes in our study was found to be a significant risk factor which predicts mortality.

TABLE XII

		SURVIVORS	%	
DIABETIC(n=30)	5(14.2)	25(71.5)	85.7	
NON DIABETIC	1(2.9)	4(11.4)	14.3	PVALUE .0144

GRAPH VIII



Preexisting chronic renal failure was found in 4 patients (11.4%).

Patients affected with HIV are a new sub group where the incidence is increasing Elem et al reported the impact of HIV virus on fournier's gangrene in Africa. He noted that in this sub group younger patients are affected and they had the gangrene process extensive and spreading to anterior abdominal wall and thigh. Fournier's gangrene had increased predisposition in second stage of disease(WHO nomenclature) where there is decreased CD 4 count.

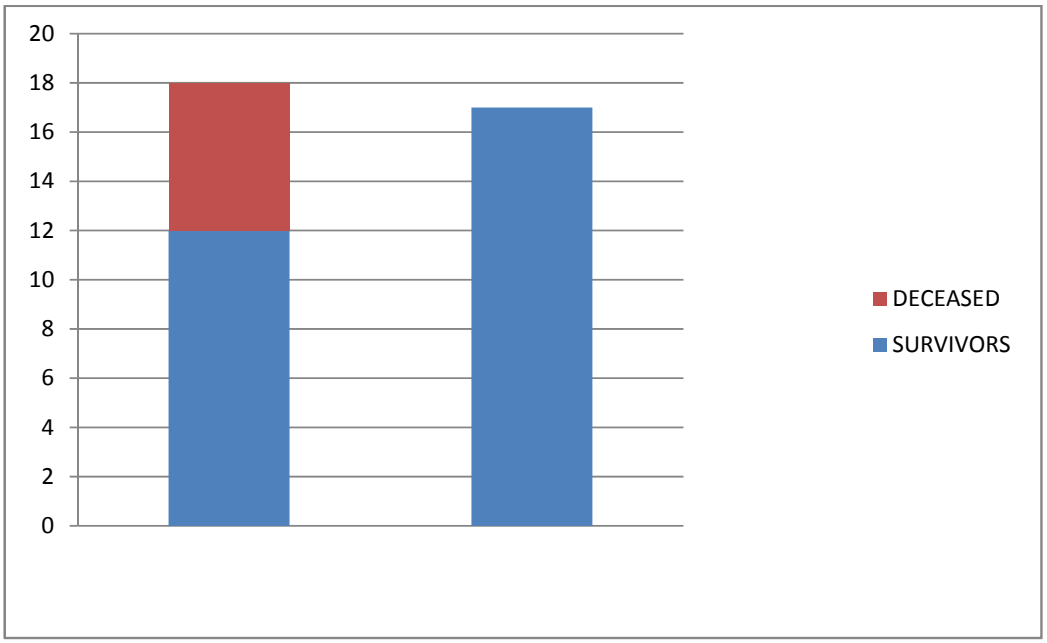
2 patients in our study had HIV seropositivity. They were aged 41 and 40 years. One belonged to upper socioeconomic class (Mech. Engineer from Sikkim) and he was only one who belonged to upper social status in the whole study. Both of them had extensive involvement beyond perineum. One patient was already receiving ART therapy during presentation. His CD4 count was 375/mm3. Other patient CD 4 count (298 / mm3) was done after admission and he was started on ART.

When more than two co morbidities are found in same patient it was found to have a significant impact on survival (Corcoran et al, basoglu et al and Yanar h et al).In our study more than two co morbid illness was found in 18 subjects. All patients (6/35) who died had greater than 2 co morbid illness.

TABLE XIII

	SURVIVORS	DECEASED	
>2 COMORBID ILLNESS(n=18)	12	6	
< 2 COMORBID ILLNESS (n=17)	17	0	P VALUE .0303

GRAPH IX



ETIOLOGY

Though the initial description of disease was of idiopathic nature the immediate etiological agent was found in about 70% of cases in most series. Eke et al reported that about 36% of cases had no source of infection to precipitate the disease process.

In our study 34.3% of cases had no recognizable source of infection. In literature the colorectal source was the most common source of infection and in our study it was 42.8%.

Unalp and Kim Yong et al proved that colorectal source of infection had increased mortality rate than other sources of sepsis. In our study 50 %(3/6) of non survivors had colorectal source of infection. Since majority of the disease was due to colorectal etiological causes there is probability that it is increasingly associated with mortality. Also the infection from urogenital region is initially limited by perineal membrane but the perianal infection and gangrene easily spreads to anterior abdominal wall.

DURATION OF SYMPTOMS

The range of duration of symptoms of was from 2 to 7 days. Yeniyol et al found that duration of symptoms was a significant factor affecting mortality. None of the patients who presented before 48 hours had death as outcome. Unalp et al ,Yeniyol et al , Kim et al and Akgun et al found that duration of symptoms >3 days was associated with increased mortality. In the study we found that the mean of all patients with mortality was found to be 4.58 and this increased duration was found to be statistically significant.

Variable	MORTALITY	N	Mean	Std. Dev	P-Value
DURATION OF SYMPTOMS (DAYS)	No	29	2.66	0.877	<0.001
	Yes	6	4.5	0.801	

Fournier's gangrene severity index

When first introduced in 1995 by Laor et al he found that the mean score of all non survivors was found to be 13.5. Subsequently in studies conducted by Yeniyol,Ulu,Cyzmet,Erol,Lujan[49] the results are given below.

TABLE XIV

AUTHOR	MEAN FGSI SCORE SURVIVORS	MEAN FGSI SCORE NON SURVIVORS
LAOR /1995	6.9 ± 0.9	13.5 ± 1.5
YENIYOL/ 2004	3.0± 1.8	12 ± 2.4
ULU / 2009	5.04 ± 2.49	13.6 ± 4.61
CYZMET/ 2009	5 ±2.9	13.5 ± 2.62
EROL/2010	5± 2.91	13.61 ± 4.7
LUJAN /2010	6.7± 0.14	9.7
Unalp et al 2010	5.4 ±0.12	11.2

In most of the studies the ROC curve was plotted and the best probable score for predicting the mortality was found out. In all studies quoted above, FGSI score greater 9 was highly predictive of mortality

In our study the mean age of deceased was 14.5. Using ROC plot curve the best cut off score for mortality was found to be 11. The higher mean score in our patients was due to the delayed presentation after systemic sepsis has set in.

The sensitivity and specificity of the score 11 to predict the non survival status was 100%.

A FGSI Score greater than 9 was defined to have higher mortality. In our study we had 3 patients with score greater than 9 (9, 10 &10) who had survived. This highlights the importance of more aggressive management in this group of patients which offers them a survival benefit. In our study, patients with score greater than 9 had an average of 3 debridement to control the spread of gangrene and infective process. This debridement was done under anesthesia besides the regular dressing and debridement in wards.

Chawla[50] in 2003 suggested in his study that no. of debridements were found significantly higher in survivors. This was explained by the fact that sicker patient who had survived the initial attack would require multiple debridements to limit the disease process .

Ersay et al[51] noted that subjects with higher FGSI score had increased no. of debridement and increased duration of hospital stay. In other study by Cemal Goktas[52] FGSI score was made before each debridement. He found that a higher score before each debridement predicted the possibility of multiple debridements at a future date. This was explained by the fact that cases where the score was increasing the disease and septicemia was also

progressing. So they required further debridements to control the disease process. To conclude the FGSI score also predicts the morbidity in survived patients. They require multiple debridements and possibly a diverting procedure to limit the contamination.

CONCLUSIONS

Fournier gangrene is a notorious surgical emergency which requires early diagnosis and treatment.

Minor infection in predisposed individuals should be given due attention and treated promptly as negligence may lead to this life threatening complication. Proper education should given to these individuals regarding warning symptoms and they should be advised to seek medical help if any signs appear.

Once diagnosed, early stabilization of hemodynamic status and immediate debridement of whole necrotic tissues with appropriate antibiotic cover will certainly reduce the risk of morbidity.

Presence of Diabetes, advanced age, primary colorectal source of infection, delayed presentation above 48 hours , systemic sepsis on admission are individual risk factors described to predict the mortality.

FGSI score is a simple score based on vital parameters and basic lab investigations.

A FGSI score greater than 9 should alert the surgeon to carry out immediate and extensive surgical debridement. This offers these patients a sure survival benefit.

A score greater than nine also predicts the morbidity. Patients with score greater than 9 who survived will probably have extensive area of involvement. They will need multiple surgical debridements and diversion procedure to hasten wound healing.

Hence it is very useful and simple test which can be easily reproduced and aids in both prediction of mortality and morbidity.

BIBLIOGRAPHY

1. Litchfield WR. The bitter sweet demise of Herod the Great. J R Soc Med 1998; 91: 283-4.

2. Fournier J-A. Gangrene foudroyante de la verge. Semaine Medicale 1883; 3: 345-8.

3. Meleney FL. Hemolytic streptococcus gangrene. Arch Surg 1924; 9: 317---64.

4. Wilson B. Necrotizing fasciitis. Am Surg 1952; 18: 416-31.

5. Gray JA Gangrene of the genitalia as seen in advanced periurethral extravasation with phlegmon. J Uro11960; 84: 740-5.

6. Luckett WH. Large phagedenic ulcer of abdomen. Ann Surg 1909; 50: 605-8.

7. Bubrick MP, Hitchcock CR. N ecrotizing anorectal and perineal infections. Surgery 1979; 86: 655-62.

8. Smith GL, Bunker CB, Dinneen MD. Fournier's gangrene. Br J Urol 1998; 81: 347-55.

9. Adeyokunnu AA. Foumier's syndrome in infants. A review of cases from Ibadan, Nigeria. Glin Pediatr 1983; 22: 101-3.

10. AdamsJRJr, MataJA, BocchiniJA, Venable DD, Culkin DJ. Foumier's gangrene in children. Urology 1990

11. Roberts DB, Hester LLJr. Progressive synergistic bacterial gangrene arising from abscesses o f the vulva and Bartholin's gland duct. Am J Obstet Gynecol1972; 114: 285-91.

12. Addison W A, Livengood CH m, Hill GB, Sutton GP, Fortier KJ. Necrotizing fasciitis of vulvar origin in diabetic patients. Obstet Gynecol1984; 63: 473-9.

13. LowthianJT, Gillard LJ Jr. Postpartum necrotizing fasciitis. Obstet Gynecol1980; 56: 661-3.

14. N.EKE, Fournier gangrene review of 1726 cases; British Journal of Surgery 2000,87,718-728.

15. Rajbhandari SM, Wilson RM. Unusual infections in diabetes. Diabetes Res Clin Pract 1998; 39: 123-8.

16. Baskin LS, Carrol PR. Necrotizing soft tissue infections of the perineum and genitalia. ***Br J Urol*** 1990; 65: 524-529.

17. Gaeta M, Volta S, Minutoli A, Bartiromo G, Pandolfo l. Fournier gangrene caused by a perforated retroperitoneal appendix: CT demonstration. AJR Am J Roentgenol1991; 156: 341-2.

18. Eke N. Colorectal cancer presenting as Fournier's gangrene. Am J Gastroenterol1999; 94: 858-9 (Letter).

19. Gould SWT, Banwell P, Glazer G. Perforated colonic carcinoma presenting as epididymo-orchitis and Fournier's gangrene. Eur J Surg Onco11997; 23: 367-8.

20. Klutke CG, Miles BJ, Obeid F. Unusual presentation of sigmoid diverticulitis as an acute scrotum. J Uro11988; 139: 380-1

21. Fialkov JM, Watkins K, Fallon B, Kealey GP. Fournier's gangrene with an unusual urologic etiology. Urology 1998; 52: 324-7.

22. CamposJA, MartosJA, Gutierrez del Pozo R, Carretero P. Synchronous cavemo-spongious thrombosis and Fournier's gangrene. Arch &Esp Uro11990; 43: 423-6.

23. Lena ekelius, Hilding, bjorkman,. Fournier's Gangrene after Genital Piercing Scand J Infect Dis 36: 610_/612, 2004.

24. Adeyokunnu AA. Foumier's syndrome in infants. A review of cases from Ibadan, Nigeria. Glin Pediatr 1983; 22: 101-3.

25. CIar LD 1lI, WhiteJJ Jr, DavidsonJT, ChandlerJJ. Early recognition and successful management of pelvic cellulitis following hemorrhoidal banding. Dis Colon Rectum 1986; 29:579-81.

26. Vastyan A, Gulacsy 1, F azekas Z. F ournier gangrene following prostatic puncture. Oro Heti11994; 135': 2039-40.

27. Moreira CA, Wongpakdee S, Gennaro ARo A foreign body (chicken bone) in the rectum causing extensive perirectal and scrotal abscess: report of a case. Dis Colon Rectum 1975; 18: 407-9.

28. BENIZRI E., FABIANI P., MIGLIORI G. *et al.* Gangrene of the perineum. *Urology*, 1996, **47** : 935-44.

29. YANG S. C., WU T. J. Fournier's gangrene : Taiwan experience. *Chin Med J (Tai pei)*, 2001, **64** : 239-243.

30. Ben Paty R, Smith AD. Gangrene and Fournier's gangrene. *Urol. Clin. North Am.* 1992; **19** : 149–62

31. Aharon U, Borenstein A, Eisenkraft S, Lifschitz O, Leviav A. Extensive necrotizing soft tissue infection of the perineum. Isr J Med Sci 1996; 32:745-749.

32. MCLATCHIE G. R., LEAPER D. J. eds. Oxford Handbook of Clinical Surgery. 2nd ed. Oxford UK : Oxford University Press, 2003 : 53,890

33. Bubrick MP, Hitchcock CR. N ecrotizing anorectal and perineal infections. Surgery 1979; 86: 655-62.

34. Gerber GS, Guss SP, Pielet RW. Foumier's gangrene secondary to intra-abdominal processes. Urology 1994; 44: 779-82.

35. Rajan DK, Scharer K. Radiology of Fournier□s gangrene. AJR Am J Roentgenol 1998;170:163–168.

36. Sherman J, SollidayM, Paraiso E, Becker J,Mydlo JH. Early CT findings of Fournier□s gangrene in a healthy male. Clin Imaging 1998;22(6):425–427

37. Laucks SS n. Fournier's gangrene. Surg Clin NorthAm 1994; 74: 1339-52.

38. SpimakJP, ResnickMI, Hampel N, Persky L. Fournier's gangrene: report of20 patients. J Uro11984; 131: 289-91.

39. Osegbe DN,Akaiso OE, PanchalingamL, Dania F, Harry A, Ashiru B. Fournier's gangrene: infective gangrene of the genitalia. Lllgos] Surg 1998; 1: 3-8

40. Adeyokunnu AA. Foumier's syndrome in infants. A review of cases from Ibadan, Nigeria. Glin Pediatr 1983; 22: 101-3.

41. Jimeno, Díaz de Brito & Parés 2010. Antibiotic tratment in Founier□s Gangrene. Cirugía Española 2010;88(5):347-351 .

42. Akcan A, Sözüer E, Akyildiz H, Yilmaz N, Küçük C & Ok E. (2009) Necessity of preventive colostomy for Fournier's gangrene of the anorectal region. Ulus Travma Acil Cerrahi Derg. 2009 Jul;15(4):342-6.

43. Capelli-Schellpfeffer M, Gerber GS. The use of hyperbaric oxygen in urology.J Urol 1999;162:647–54

44. Barkel DC, Villalba MR. A reappraisal of surgical management in necrotizing perineal infections. Am Surg 1986; 52: 395-7

45. Smith KA. Interleukin-2: inception, impact, and implications. Science. 1988;240:1169-76.

46. Saleh El Awady, Wael Khafagy, Mohamed Abd El-Raof, Elham ragab FOURNIER'S GANGRENE: Clinical, Biochemical, Bacteriologic, Immunologic study and Treatment outcome Egyptian Journal of Surgery Vol 26, No 2, April, 200

47. Cuccia G, Mucciardi G, Morgia G, d'Alcontres FS, Gali A, Cotrufo S,Romeo M, Magno C. Vacuum-assisted closure for the treatment of Fournier's gangrene. Urologia Internationalis 2009;82:426-431.

48. Tucci G, Amabile D, Cadeddu F, Milito G. Fournier's gangrene wound therapy: our experience using VAC device. Langenbecks Arch Surg 2009;394:759-760.

49. Yeniyol CO, Suelozgen T, Arslan M, Ayder AR. Fournier's gangrene: experience with 25 patients and use of Fournier's gangrene severity index score. Urology 2004;64:218-22.

50. Chawla SN, Gallop C, Mydlo JH. Fournier's gangrene: An analysis of repeated surgical debridement. Eur Urol 2003;43:572-5.

51. Cemal Goktas, Mehmet Yildirim Factors Affecting The Number Of Debridements In Fournier's Gangrene: Our Results in 36 Cases; Turkish Journal of Trauma & Emergency Surgery Cit - Vol. 18 - No. 1, 45-47.

ANNEXURES

PROFORMA

OBSERVATIONAL STUDY OF FOURNIER'S GANGRENE AND USEFULNESS OF FOURNIER'S GANGRENE SEVERITY INDEX IN PREDICTING THE OUTCOME

PATIENT DATA:

PATIENT NAME : AGE/SEX:

Hospital Number :

Date of admission :

Date of surgery :

Date of discharge :

History of illness :

i. Presenting complaints:

ii. Disease onset

iii. Duration of symptoms

iv. Progress of symptoms

Predisposing factors:

i. Diabetes
ii. Alcoholism
iii. Malignancy
iv. Immune compromised state
v. Morbid obesity
vi. Low socioeconomic status

Causative factor:

- Anorectal
- Urogenital
- Cutaneous
- Idiopathic

Patient examination:

Vital parameters :

Parameter	**Patient's value**	**Score allotted**
Heart rate		
Respiratory rate		
Temperature		

Blood investigations:

Parameter	**Patient's value**	**Score allotted**
Hematocrit		
Leukocyte count		
Creatinine		
Serum sodium		
Serum potassium		
Serum bicarbonate		

FOURNIER GANGRENE SEVERITY INDEX SCORE:

Extent of the disease:

Wound culture and sensitivity:

Details of debridement and other surgeries:

Reconstruction procedure used:

Total duration of hospital stay :

Number of surgeries performed :

Follow up :

OBSERVATIONAL STUDY OF FOURNIER'S GANGRENE AND USEFULNESS OF FOURNIER'S GANGRENE SEVERITY INDEX IN PREDICTING THE OUTCOME

Investigator: **Dr. P .NAVEEN**, II yr Post Graduate MS (Gen Sur)

Guide: **Prof. Dr. A. RAJENDRAN,** Chief, Surgical Unit 6, Govt. Stanley Hospital.

Informed Consent

Name: Age/ Sex: IP:

I herewith declare that I have been explained in a language fully understood by me regarding the purpose of this study, methodology, details and data collected for this study.

I have been given an opportunity to discuss my doubts and I have received the appropriate explanation.

I understand that my participation in this study is completely voluntary and that I am free to withdraw from this study at anytime without any prior notice &/ or without having my medical or legal rights affected.

I permit the author and the research team full access to all my records at any point, even if I have withdrawn from the study. However my identity will not be revealed to any third party or publication.

I, being a person of sound mind, hereby give my permission to use a photograph(s) of me taken intra operatively in the dissertation.

I herewith permit the author and the research team to use the results and conclusions arising from this study for any academic purpose, including but not limited to dissertation/ thesis or publication or presentation in any level.

Therefore, in my full conscience, I give consent to be included in the study and to undergo any investigation therein.

Patient's Sign Investigator's Sign

When completed, 1 for patient, 1 for researcher site file, 1 (original) to be kept in medical notes

OBSERVATIONAL STUDY OF FOURNIER'S GANGRENE AND USEFULNESS OF FOURNIER'S GANGRENE SEVERITY INDEX IN PREDICTING THE OUTCOME

Investigator: **Dr. P .NAVEEN**, II yr Post Graduate MS (Gen Sur)
Guide: **Prof. Dr. A. RAJENDRAN,** Chief, Surgical Unit 6, Govt. Stanley Hospital.

PATIENT INFORMATION MODULE

Invitation to Participate:

You are being invited to participate in this study. Since you have been diagnosed to have Fournier's gangrene you are being invited to participate in this study. You have the right to know that consent is purely voluntary, that your permission to participate in research may be withdrawn at any time, and that you may discontinue participation in all or part of the research without affecting your present or future medical care. Please take your time to review this form. Feel free to discuss it with your family before you make your decision.

What is Fournier's gangrene and what is the nature and course of the disease?

Fournier's gangrene is a necrotizing soft tissue infection involving the scrotum and perineal area. This gangrene involves the skin and subcutaneous tissues initially. If these gangrenous and infected areas are not surgically removed immediately it will lead to rapid spread to abdominal wall and chest wall and death due to sepsis and multi organ failure.

Why is This Study being done and aim of the study?

This study analyses the age distribution, predisposing factors and risk factors of patients diagnosed with Fournier's gangrene.
The study observes the need for surgical debridement and other procedures in management of Fournier's gangrene
To assess the usefulness of an index called Fournier's gangrene severity index which uses simple parameters to predict the mortality rate.
.

How Many People Will Take Part in the Study?

About 30 patients are expected to take part in this study. All patients diagnosed with Fournier's gangrene between the ages 20-80 will be included in the study.

What are the details obtained from the patient and investigation done in this Study?

If you agree to participate in this study, patient or relatives will be asked to sign the consent form.
This study is primarily done to assess the patient's disease severity and mortality rate. Once the patient is diagnosed as a case of Fournier's gangrene and admitted to emergency ward of Govt. Stanley hospital the patient's history, predisposing factors and risk factors are noted down.
Patient's heart rate respiratory rate and body temperature are noted down. Later the routine blood investigations are done. The Fournier's severity index score is calculated based on the above parameters.

Does the modality of treatment received by the patient change in this study?

This study observes the predisposing factors, risk factors and predicts the mortality using the Fournier severity index score. In this study the treatment modality of immediate surgical debridement and further wound care remains the same and no new form of treatment is being instituted. There will be no delay in the initiation or progress of treatment due to the study.

What Are the Risks of the Study?

There are virtually no risks involved in the study. The vital parameters and blood investigations of the patients are used. When blood is requested, a trained technician, nurse or doctor will obtain the blood samples. Complications that may arise as a result of blood collection are typically minimal and may include bruising and swelling.

The mode of treatment, principles of surgery, dressings and wound care of patients with Fournier's gangrene are adhered to standard protocols and no modifications are done to these. Since it's an observational study the natural

course of disease and hospital data regarding surgery, wound care and final outcome of the patients are observed, noted and stastically analysed.

There will not be any monetary benefits for participating in this study.

Are There Alternatives to the Study?
You have the alternative of not participating in this study. Your participation is completely voluntary and will not affect your medical treatment now or in the future.

All information collected in this study will be kept strictly confidential. The information regarding the disease and lab details obtained from the patient in this study will be used for statistical analysis and research purposes. The patient's age, sex and other personal information will not be revealed. The data collected and the Fournier gangrene severity index score will be used to predict the outcome and results will be stastically analysed.

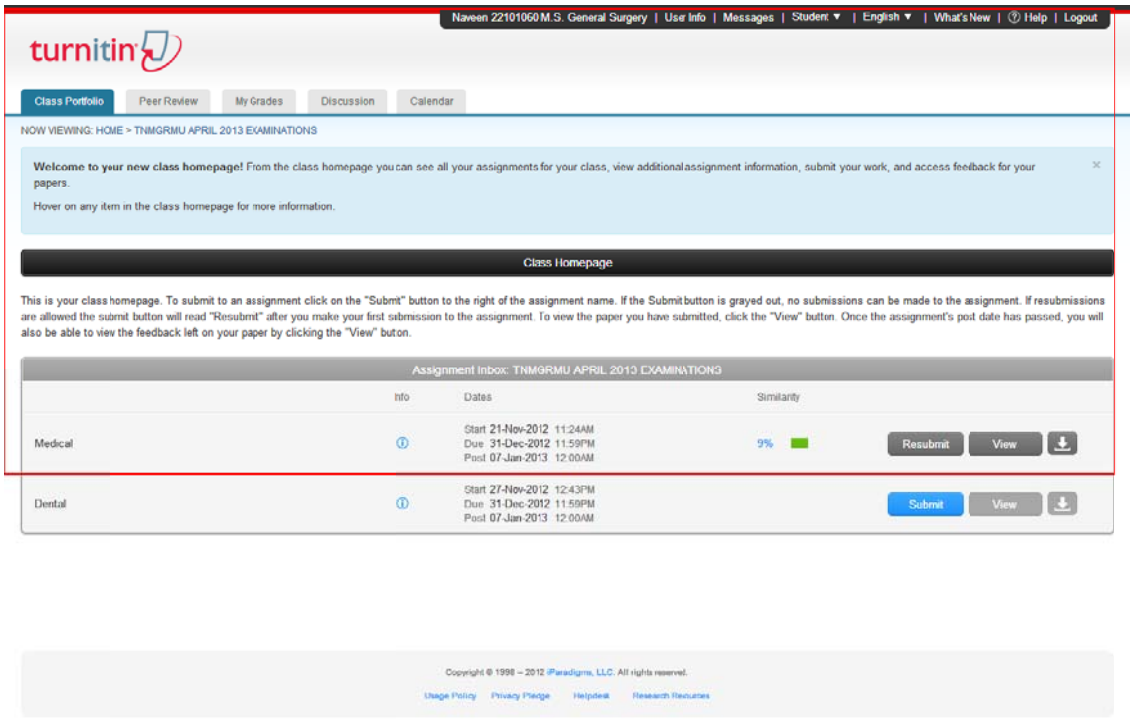
Investigator's Sign

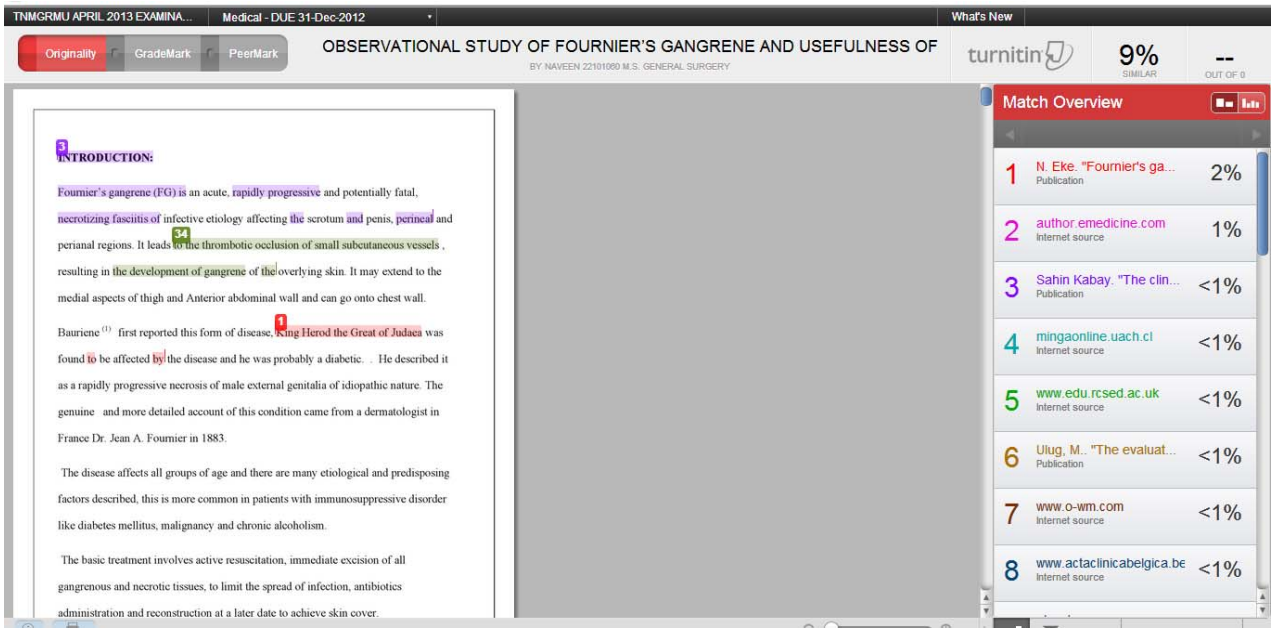
Patient's Sign
Name of the patient

Date :

MASTER CHART

NAME	AGE	SEX	IP NO.	PRE DISPOSING FACTORS	ETIOLOGY	DURATION OF SYMPTOMS(DAYS)	TEMP	HEART RATE	RR	HEAMTOCRIT	TOTAL COUNT	CREATININE	SODIUM	POTASSIUM	BICARBONATE	FGSI SCORE	NO. OF DEBRIDEME-NTS	RENAL FAILURE	MORTALITY
MOHAN	62	M	2319	DM	CR	3.5	38.5	98	20	42	9000	1.9	138	3.9	22	2	1	YES	NO
RAJAN	62	M	2923	DM/Ch.A	CR	4	39	118	36	40	16,000	3.2	127	2.8	26	12	2	YES	YES
ELLAPAN	50	M	598	DM	CR	3.5	38.6	104	26	42	11,000	1.7	139	3.6	24	3	1	YES	NO
VENKATESAN	74		432	DM/A	CR	4	38.4	98	20	44	10500	2.1	139	4.1	28	3	1	YES	NO
PERUMAL	51	M	1278	DM/A	UG	2	39.12	112	22	46	3800	1.8	126	5.2	31	8	1	NO	NO
KRISHNAN	48	M	2034	NONE	CR	2	39.1	112	29	36	12000	1.2	132	3.6	29	6	1	NO	NO
KRISHNA MOORTHY	53	M	2343	CKD	UG	3	38.6	114	30	48	11200	2.7	138	5.1	20	7	1	YES	NO
RAMASAMY	78	M	2654	DM/Ch.A/CKD	CR	5	39.2	114	28	32	13,500	3.8	129	5.2	32	16	1	YES	YES
KRISHNAMOORTHY	68	M	2964	DM/HF/Ch.A	CR	4.5	39.2	128	26	36	20,500	3.8	128	3.2	28	19	1	YES	YES
RAMAPOORANAM	51	M	2835	DM	CR	2	38.6	96	26	42	13600	1.2	138	3.9	26	2	2	YES	NO
RAMAKRISHNAN	71	M	3245	DM/HF	CUT	3	38.3	106	22	38	10800	1.3	129	4.5	28	1	2	YES	NO
KUMAR	68	M	4309	DM	ID	2	38.4	108	24	39	8600	1.6	136	4.2	30	2	1	YES	NO
MURUGAN	41	M	6432	DM/Ch.A/HIV	UG	2.5	37.1	96	23	43	43	7600	136	3.8	23	0	1	NO	NO
KAMALESHWARAN	66	M	7493	DM/Ch.A	CUT	4.5	39.1	112	26	44	14000	1.6	138	4.1	21	10	3	YES	NO
PALANI	53	M	8634	DM/Ch.A	ID	2	37.6	106	35	48	12000	1.2	141	4.3	26	1	2	YES	NO
DASAN	52	M	12321	DM/Ch.A	CR	2	38	112	26	44	13800	1.4	141	3.9	20	3	2	YES	NO
SUMATHI	46	F	12654	DM	ID	2	37.6	102	36	48	7900	1.1	139	3	29	2	2	YES	NO
JOSEPH	66	M	13421	DM	ID	4	37.6	98	26	48	1400	1.4	133	4.1	26	2	1	YES	NO
SALEEM	40	M	18345	HIV	CUT	3	39.1	108	26	44	2800	1.7	128	4.1	23	10	4	NO	NO
CHINNAYIAN	49	M	20346	DM/Ch.A	ID	1	39.1	102	21	42	12,000	0.8	137	3.2	22	4	2	NO	NO
MOTIYAN	64	M	23674	DM/Ch.A/CKD	ID	4	38.6	98	26	46	16000	3.2	128	5	19	11	1	YES	YES
JABBAR ALI	47	M	27432	DM/Ch.A	ID	2	38	102	28	40	12500	1.2	139	4.8	18	3	1	NO	NO
ISSAC	54	M	29321	DM/Ch.A	ID	3	38.6	108	26	42	13000	1.9	142	4.1	24	4	1	YES	NO
PARANTHAMAN	41	M	32120	NONE	CR	2.5	38.7	118	22	39	13500	1.4	137	4.2	28	3	1	NO	NO
SENGUTUVAN	54	M	33400	DM	CR	2	37.9	98	24	42	10,500	1.9	139	4.2	29	2	1	YES	NO
PURUSHOTHAMAN	48	M	35432	DM	ID	2	37.8	100	28	40	9000	1.2	128	4.1	18	4	1	NO	NO
KANNAN	76	M	37823	DM/HF	CR	4	39.2	108	30	44	8000	1.9	128	4.9	19	10	3	YES	NO
PARTHIBAN	61	M	40193	CA/Ch.A	CUT	6	39.1	114	26	46	16,500	2.2	125	4.1	21	13	1	YES	YES
KRISHNAN	59	M	42156	DM	ID	2.5	37.2	118	26	42	10800	1.9	136	4.8	19	4	2	YES	NO
SADIYANDI	55	M	47321	DM	ID	2.5	37.9	98	22	39	9800	1.6	139	3.9	18	2	1	YES	NO
SRIKANTH	62	M	49354	DM	CR	4	37.8	98	24	44	10200	1.8	141	3.8	23	2	1	YES	NO
RAJENDRAN	58	M	53219	DM	CR	3	37.6	96	22	48	9900	1.9	138	4.8	19	4	2	YES	NO
JAMES VICTOR	47	M	56783	DM/Ch.A	ID	2	37.2	98	28	48	10800	1.2	139	4.9	23	2	1	NO	NO
MITHLESH	44	M		DM	CR	1.5	38.2	110	24	43	9800	1.3	138	3.8	25	2	1	NO	NO
VENKATESHWARAN	62	M	58732	DM/Ch.A/CKD	UG	4	38.6	112	20	48.2	16,300	3.2	128	3	20	13	1	YES	YES

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OBSERVATIONAL STUDY OF FOURNIER'S GANGRENE AND USEFULNESS OF
BY NAVEEN 22101060 M.S. GENERAL SURGERY
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INTRODUCTION:
Fournier's gangrene (FG) is an acute, rapidly progressive and potentially fatal, necrotizing fasciitis of infective etiology affecting the scrotum and penis, perineal and perianal regions. It leads to the thrombotic occlusion of small subcutaneous vessels , resulting in the development of gangrene of the overlying skin. It may extend to the medial aspects of thigh and Anterior abdominal wall and can go onto chest wall.
Baurienne (1) first reported this form of disease, King Herod the Great of Judaea was found to be affected by the disease and he was probably a diabetic. . He described it as a rapidly progressive necrosis of male external genitalia of idiopathic nature. The genuine and more detailed account of this condition came from a dermatologist in France Dr. Jean A. Fournier in 1883.
The disease affects all groups of age and there are many etiological and predisposing factors described, this is more common in patients with immunosuppressive disorder like diabetes mellitus, malignancy and chronic alcoholism.
The basic treatment involves active resuscitation, immediate excision of all gangrenous and necrotic tissues, to limit the spread of infection, antibiotics administration and reconstruction at a later date to achieve skin cover.
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